THE ROLE OF AGE IN THE SYMPTOMATIC EXPRESSION OF DEPRESSION, COMORBIDITY AND THE OCCURRENCE OF IDENTIFIED RISK FACTORS: A STUDY OF EARLY AND MIDDLE ADULTHOOD

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ABSTRACT

A controversial topic in the literature on depression is the extent to which childhood, adolescent and adult depression are the same disorder. Surprisingly, the extent to which young and middle adult depression are similar disorders has not specifically been researched. This study analysed data from 431 participants, aged 18 to 54, who meet DSM-III-R diagnostic criteria for a MDE. Data came from three large studies conducted by the Department of Psychological Medicine. Baseline data from multiple measures of symptomatology, comorbidity, and identified risk factors were analysed to investigate age-related differences in the occurrence of these factors. Results of the study suggested that symptomatology, comorbidity and the occurrence of identified risk factors change during young and middle adulthood. Many of the symptoms common in young adults were similar to those suggested in the literature to be common in adolescents. This study highlights the significance of developmental differences in depression during adulthood and cautions that combining 'adults' into one bracket may disguise critical differences. It was also observed that developmental trends were present on some measures but not others suggestive of an age-related bias between self-report measures and clinician rated scales.

CHAPTER 1

INTRODUCTION

1.1 LITERATURE REVIEW

Major Depressive Disorder (MDD) is a recurrent episodic disorder that occurs during childhood, adolescence, and adulthood. Historically, it has been thought that depression was rare during childhood. However, childhood depression has become an established area of research and although there is debate as to its similarity to adult depression, generally it is agreed that childhood depression is not as rare as once thought. Although, MDD does present during childhood, it appears that the most common age of onset occurs during adolescence or young adulthood. A community sample studied by Lewinsohn, Clark, Seeley, and Rohde (1994) reported the mean age at onset of first depressive episode was approximately 15 years. However, Weissman, et al. (1996) found that the mean age at onset ranged from 25 to 35 years across 10 countries. Overall, it is well documented that during adolescence, there is an increase in depressive symptomatology when compared to childhood (Rutter, 1986; Angold, 1988a; Angold, 1993) and that once MDD arises it is a recurrent disorder (see Kovacs, 1996). This has led researchers to investigate differences across the lifespan, particularly differences between child-, adolescent-, adult- and older adult-onset depression, and the continuity of MDD over the lifespan. Surprisingly, differences between early and middle adulthood have not been addressed.

1.1.1 Continuity, Relapse and Recurrence

As mentioned, depression is considered to be a recurrent, episodic disorder. It has been reported that the median time to recovery from MDD for children and adolescents is shorter (7-9 months) than that of adults (12 months). In addition, recovery from an initial MDD episode is shorter than the recovery from subsequent episodes (Kovacs, 1996). Longitudinal studies of the course of childhood-, adolescent- and adult-onset MDD suggest that the recurrence rate of MDD is substantial despite differences in rates. Some research has shown that over seven years, up to 90% of initially clinically referred, depressed, adult patients have at least one recurrent episode of MDD and about 70% of children and adolescents have a recurrent MDD episode over five years (see Kovacs, 1996). In addition, children with post-pubescent onset MDD appear to be at a greater risk of recurrence than children with pre-pubescent onset MDD (Harrington, Fudge, Rutter, Pickles, & Hill, 1990).

Other research has demonstrated that in a community sample of 19 to 24 year old individuals with a history of adolescent MDD, 45% experienced a recurrence of MDD in young adulthood (Lewinsohn, Rohde, Klein, & Seeley, 1999). Harrington, et al. (1990) reported recurrence rates of depression from adolescence into adulthood were 60-70%. This research suggests that in those who have experienced a depressive disorder at any point, there is a high rate of continuity of risk for MDD into later development.

1.1.2 The Influence of Gender

A highly consistent finding within the research investigating MDD is that women are twice as likely to experience MDD than men (Joyce, 2000). Women have higher rates than men in both community and clinical samples (see Carter, Joyce, Mulder, Luty, & McKenzie, 2000). With regard to age, during childhood there is a slightly higher prevalence rate of depression in boys than girls but during puberty the increased rate in girls occurs (Joyce, 2000). Research has also reported gender differences in the manifestation of MDD. For example, women tend to present with an increase in appetite and weight and men tend to present with lower psychological, social and occupational functioning. A number of other symptoms have also been identified to present differently in men and women, however, findings have been inconsistent (see Carter, et al., 2000).

1.1.3 Definitions of Depression

The term 'depression' is often used to describe mood. For example, 'depressed mood' is familiar to most people as a description of being 'unhappy' or feeling worthless (Rippere, 1994). However, 'depression' in used in this thesis to describe a clinical disorder. Symptoms of depression can include feeling lonely, feeling the need to be perfect, feeling worthlessness, nervous, guilty, sad, worrying (Peterson, Maier, Seligman, 1993), a depressed mood, lack of energy, weeping, social withdrawal, negative cognitions and some patients experience hallucinations or delusions (Rippere, 1994). In general, depression refers to a cluster of symptoms covering changes in affect, cognition and behaviour (Rippere, 1994). According the Diagnostic and Statistical Manual of Mental Disorders, 4th Edition, Text Revision (DSM-IV-TR; American Psychiatric Association; APA, 2000), Major Depressive Disorder (MDD) is characterised by one or more Major Depressive Episodes (MDE). MDE's present as a depressed mood or anhedonia[1], and five or more of the following symptoms: appetite or

[1] Anhedonia is the loss of interest or pleasure in previously enjoyed activities.

weight loss or gain, insomnia or hypersomnia, psychomotor agitation or retardation[1], fatigue, feelings of worthlessness or guilt, poor concentration or indecisiveness, and/ or recurrent thoughts of death, suicidal ideation or suicide attempt. In children, or adolescents, an irritable mood may be more evident than a depressed mood, and failure to make expected weight gains may be more evident than weight or appetite loss or gain. In addition to these symptoms, individuals experience significant distress or impairment in social, occupational, or other important areas of functioning. Symptoms persist for most of the day, nearly everyday, for at least two consecutive weeks. One of the differential diagnoses for MDD is another mood disorder defined in the DSM-IV-TR, Dysthymic disorder. Dysthymic disorder shares similar symptoms with MDD, however, symptoms are present for more days than not over a period of at least two years.

The DSM-IV-TR (APA, 2000) criteria have been criticised because, aside from the additional criteria for children and adolescents above, it makes no distinction in the criteria between prepubescent, adolescent and adult depression. In prepubescent children with MDD, somatic complaints, agitation and mood-congruent hallucinations are said to be frequent. In adolescents, wanting to leave home, restlessness, grouchiness, and aggression are common (see Harrington, 1993). However, these qualities are not in the criteria. The criteria also do not include developmental differences in symptomatology over different phases of adulthood.

1.1.4. Epidemiology of Depression

The prevalence rate of adult MDD has been estimated to be approximately 2 to 5%, based on studies reporting 6-month, 1-month and 1-week rates. The lifetime prevalence rate is approximately 10 to 20% (Joyce, 2000). However, cultural variability has been reported, with lifetime rates ranging from 2 to 19% and annual rates ranging from 1 to 6%, across 10 countries (Weissman, et al., 1996). Christchurch data suggests that the lifetime prevalence of MDD ranges from 13 to 33% in adults, and Christchurch and Dunedin samples have shown that the lifetime prevalence rate in 15-year olds ranges from 5 to 17% (see Sullivan & Bulik, 1997). Other data reports that the prevalence rate of depression in pre-pubescent children is approximately 2% (Fleming, Offord, & Boyle, 1989; Institute of Medicine, 1989), and 5 to 8% in adolescents (Lewinsohn, et al. 1994). Some investigators have concluded that MDD

[1] Psychomotor agitation is excessive motor activity associated with a feeling of inner tension. The activity is usually non-productive and repetitious and consists of such behaviours as pacing, fidgeting, pulling of clothes and an inability to sit still. Psychomotor retardation is a visible generalised slowing of movements and speech.

occurs in older adolescents at levels comparable with levels in adults, with prevalence rates between 2-5% (Lewinsohn, et al. 1999).

1.1.5 Continuity of Symptomatology

Despite there being a general understanding within the literature as to the continuity of depression as a disorder over childhood, adolescence, and adulthood, controversy remains as to the extent and nature of continuity in the expression of depression. One theoretical stance states that depressive disorders are relatively similar, both etiologically and the way in which they present, across the lifespan. This has resulted in models of depression for adults being applied to depression in young adults, adolescents, and children. For example, as mentioned above, the DSM-IV-TR (APA, 2000) criteria for a MDE are almost identical for adults, adolescents and children. In addition, Angold (1988b) reported that children might express the full range of adult depressive symptoms by the age of 6 to 8 years, and Kovacs (1996) concluded that children and adolescents in a clinically referred sample with MDD had comparable symptom pictures as adults. In contrast, Verhulst and Koot (1995) state that despite advances in classifying depressive conditions, there is still a poor understanding of developmental differences in depression.

Many investigators have found differences in symptomatology across the lifespan suggesting significant developmental differences in depression (Ryan, et al., 1987; Carlson & Kashini, 1988; Cooper & Goodyer, 1993; Wallace & Pfohl, 1995; Garvey and Schaffer, 1994; Avenevoli, 1999). For example, Cooper and Goodyer (1993) found that in a community sample of 11 to 16 year old children, symptoms of depression changed with age. Somatic anxiety[1], hopelessness, guilt and obsessive ruminations decreased with age, while anhedonia, weight loss, suicidal acts and oppositional behaviour in school increased with age. However, Cooper and Goodyer also stated that most symptoms of depression such as depressed mood, social withdrawal, psychomotor agitation, early insomnia[2] and nihilistic ideas[3] are uninfluenced by age. Ryan, et al. (1987) studied pre-pubescent children and adolescents in a clinical setting. The authors found that the pre-pubescent children presented

[1] Somatic anxiety is characterised by the physiological concomitants of anxiety such as dry mouth, wind, indigestion, diarrhoea, cramps, belching, palpitations, headaches, hyperventilation, sighing, urinary frequency, and sweating.
[2] Early insomnia is difficulty in falling asleep.
[3] Nihilistic ideas are characterised by the rejection of all religious and moral principles.

with more somatic complaints, psychomotor agitation, separation anxiety, phobias, hallucinations, and a more depressed appearance, whereas the adolescents experienced greater anhedonia, hopelessness, hypersomnia, weight change, use of illicit drugs, and lethality of suicide attempts (though not more severe suicidal ideation and intent). There was no difference in the overall levels of depression across age. Carlson and Kashini (1988) compared preschoolers, pre-pubescent children, adolescents, and adults with depression. They concluded that suicidal ideation, depressed mood, decreased concentration and insomnia occurred consistently across all ages. However, low self-esteem, somatic complaints and a depressed appearance decreased with age, while anhedonia, diurnal variation in mood, hopelessness, psychomotor retardation, and delusions increased with age across all four age groups. Overall, there appeared to be some consistency across these studies: somatic complaints and depressed appearance declined with age, anhedonia and weight changes increased with age, and depressed mood, suicidal ideation and early insomnia occurred consistently across childhood and adolescence. Findings on many other symptoms, however, appear to be mixed.

1.1.5.1 Methodological Considerations

The reason for such mixed findings may be due to methodological differences between the studies. The measurement of depression varies between studies causing difficulty when comparing such studies. For example, Ryan, et al. (1987) assessed participants with the Kiddie-Schedule for Affective Disorders and Schizophrenia for School-aged children (K-SADS; Chambers, et al., 1985) whereas, Cooper and Goodyer (1993) assessed participants with the Diagnostic Interview Schedule for Children (DISC: Version XIII-III; Costello, Edelbrock, Kalas, Kessler, & Klaric, 1982, cited in Cooper & Goodyer, 1993). This resulted in different symptom definitions. Participants were also selected for different reasons. For example, participants in the Ryan, et al. (1987) study were referred to a specialist child and adolescent clinic, whereas, participants in the Cooper and Goodyer (1993) were recruited from various community schools. Hence, some studies used clinical populations and others community populations. Finally, some studies (e.g., Ryan, et al.) combine the results from boys and girls, which may have obscured significant differences in gender or significant age changes.

1.1.5.2 Further Research on Symptomatology

More recently, Avenevoli (1999) argued that few studies have systematically and satisfactorily contributed to the knowledge of the continuity of depression across the life course. Avenevoli's research, based on a community sample (age 8-18 years), asserted that the intensity of hopelessness, guilt, irritability and suicidal thoughts increased from childhood to adolescence. This study found that the behavioural stability of many depressive symptoms decreased during the transition from childhood to early adolescence but increased during the middle adolescent years. The author concluded that the transition from childhood to adolescence is a critical period for change in the manifestation and continuity of depressive symptoms.

Overall, this research has shown that there is some, though limited, understanding of the developmental changes in depression across childhood and adolescence. However, the transition from adolescence to adulthood and throughout adulthood has been neglected. This topic is of importance because adolescent and adult depression are often considered to be etiologically similar with similar presentations, and therefore receive similar treatment. In addition, the expression of depression across adulthood is considered to be relatively homogeneous and 'adults' are usually combined into one bracket. The remainder of this review explores the similarities and differences across adolescent and adult development in the symptomatology, comorbidity and the occurrence of identified risk factors for depression. At times research referring to childhood depression will be discussed to illustrate continuity or discontinuity. Older adulthood is not a focus of this thesis, hence the literature on depression in older adults will not be reviewed.

1.1.6 Symptomatology Across Adolescence, Early Adulthood and Middle Adulthood

Two studies that have investigated the influence of age on depressive symptomatology come from Wallace and Pfohl (1995), and Garvey and Schaffer (1994). Wallace and Pfohl (1995) investigated the age differences in symptomatology across adulthood in a clinical sample (18-81 years), using the Hamilton Depression Rating Scale- 24 item (HDRS-24; Hamilton, 1960, cited in Wallace & Pfohl, 1995). The total score had no association with age indicating that overall depression severity did not change with age. In both men and women, feelings of guilt and suicidal thoughts declined with age, whereas hypochondriasis and diurnal variation of mood in the morning increased. A sense of worthlessness declined with age in men, as did diurnal variation in mood in the afternoon

decline with age in women. Endorsement of the psychic anxiety [1], work and activities, psychomotor agitation, insight and feelings of helplessness items all increased with age in women. Depressed mood, insomnia, psychomotor retardation, somatic anxiety, somatic symptoms, weight loss, insight, depersonalization, paranoia and obsessions and compulsions did not show an association with age. This study indicated that an interaction between gender and age occurred for some symptoms.

Garvey and Schaffer (1994) reported that in inpatients (14-80 years) with depression, those younger than 40, were more likely to experience hypersomnia, appetite and weight gain, decreased libido, headaches, diurnal variation with worsening of mood in the afternoon and a mood characterised as sad but not anxious, whereas, those older than 40 were more likely to experience late insomnia[2] , psychomotor agitation, and diurnal variation with lower mood in the morning. However, early and middle[3] insomnia, appetite and weight loss, decreased concentration and memory, indecisiveness, hopelessness, crying, suicidal thoughts and attempts, anhedonia, loss of energy, psychomotor retardation, irritability, psychic anxiety, somatic anxiety, somatic complaints, and psychosis were not associated with age.

Research findings regarding specific symptoms of depression during adolescence and adulthood will now be reviewed.

1.1.6.1 Depressed Mood and Anhedonia

Important symptoms for a diagnosis of a MDE according to the DSM-IV-TR (APA, 2000) are depressed mood and anhedonia. As mentioned above, the occurrence of depressed mood as a symptom of MDE's appears to be uninfluenced by age (Carlson & Kashini, 1988; Cooper & Goodyer, 1993; Wallace & Pfohl, 1995). However, anhedonia does show an association with age. Anhdeonia is more common in adolescents and adults than children (Ryan, et al., 1987; Carlson & Kashini, 1988; Cooper & Goodyer, 1993), yet, anhedonia is not influenced by age in adulthood (Garvey and Schaffer, 1994; Wallace and Pfohl, 1995). According to these studies, depressed mood and anhedonia appear to occur consistently across the lifespan, except anhedonia occurs less frequently in children.

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[1] Psychic anxiety is characterised by feeling tense and restless.
[2] Late insomnia is awakening before one's usual waking time and being unable to return to sleep.
[3] Middle insomnia is awakening during the night followed by eventually falling asleep with difficulty.

1.1.6.2 Anxiety Symptoms

Anxiety is a well-research symptom of depression and it is expressed in depression in a number of ways. *Somatic* anxiety (Cooper & Goodyer, 1993), and *separation* anxiety (Ryan et al., 1987) are less common in adolescents than childhood. Some research claims that *psychic* anxiety is more common with age in adult women (Wallace & Pfohl, 1995). However, others have not supported this (Garvey & Schaffer, 1994). More consistent research reports that *somatic* anxiety does not vary across adulthood (Garvey & Schaffer, 1994; Wallace and Pfohl, 1995). Although research findings are mixed, anxiety is reported to be occur across the lifespan, however, it appears that it is expressed differently at different ages. The comorbidity of depression with anxiety *disorders* is discussed below.

1.1.6.3 Irritability

Based on the DSM-IV-TR (APA, 2000), irritability may be characteristic of child and adolescent depression but it is not a criterion for adult depression. Some research shows that irritability is experienced more in adolescents than children with depression (Avenevoli, 1999). One study demonstrated that within an adult sample, irritability declined with age in women (Wallace & Pfohl, 1995). Not all studies report this, Garvey and Schaffer (1994) concluded that irritability occurred consistently across adulthood. The findings regarding irritability in adulthood appear mixed.

1.1.6.4 Psychosis

Some investigators report that the occurrence of depression with delusions appears to be infrequent in children and adolescents (Ryan et al., 1987). Others explain that psychotic depression in children tends to present as auditory hallucinations instead of delusions as seen in adolescents and adults (Ryan et al., 1987; Birmaher, et al., 1996). Carlson and Kashini (1988) also demonstrated that the occurrence of delusions increases with age. Other research has shown that the occurrence of psychosis does not differ across adulthood (Garvey & Schaffer, 1994; Wallace & Pfohl, 1995). Therefore, while delusions may become more apparent with age, overall occurrences of psychosis do not appear to differ across adulthood.

1.1.6.5 Suicidal Behaviour, Ideation and Self-Harm

Developmental differences in suicidal behaviour and ideation has received a lot of attention within the psychological literature. The one-year prevalence rate of adolescent suicide is 2 to 6% and the lifetime prevalence rate is 3 to 7% (Birmaher, et al., 1996; Blair-

West, Cantor, Mellsop, & Eyeson-Annan, 1999). Blair-West, et al., (1999) found that 7% of males who develop depression ultimately suicide, whereas only 1% of females complete suicide. The male: female ratio for suicide risk in MDD is 10:1 for those under 25, and 5.6:1 in adults (Blair-West, et al., 1999) due to a lower rate of female youth suicide. Note, though, that within a Christchurch sample (Fergusson, Woodward, & Horwood, 2000), females reported consistently higher rates of suicidal ideation and attempts across all ages.

Estimates of lifetime suicide *attempt* rates are approximately between 10% for adults and 14% for adolescents (see Malone, Haas, Sweeney, & Mann, 1995) and the point prevalence of suicidal *ideation* (in those 15-21 years) is 29% (Fergusson, et al., 2000). A study investigating the clinical outcomes of adolescent-onset depression into adulthood found a 5-fold (26% versus 5%) increased risk of first suicide attempt when compared to those without a psychiatric illness and 22% had made multiple attempts (Weissman, et al., 1999). This highlights the high risk of attempts in young people experiencing depression. Both the frequency of suicide attempts and the lethality of attempts has been shown to increase from childhood to adolescence (Cooper & Goodyer, 1993) but the frequency of attempts was reported to be stable across age during adulthood (Garvey & Schaffer, 1994). With regard to ideation, Carlson and Kashini (1988) demonstrated that suicidal ideation occurred consistently across all ages. Not all studies support this, Garvey and Schaffer (1994) found no association with age across adulthood, Avenevoli (1999) concluded that ideation was more common in adolescence than childhood, and Wallace and Pfohl (1995) found that ideation decreased with age across adulthood.

High-Risk Periods for Attempted Suicide

One explanation for the high rates of suicidal behaviour during adolescence comes from a study by Malone, et al. (1995). Malone, et al., revealed that the first three months after the onset of an MDE and the first five years after the lifetime onset of MDD, represented the highest-risk period for attempted suicide in a clinical sample, independent of the severity or duration of depression in those 18 to 80 years. Vieta, Nieto, Gasto, and Cirera (1992) also found that most depressed patients who made a suicide attempt, did so within the first 12 months of the depressive episode. Adolescence is a common time of onset for MDE's, and thus perhaps a high-risk time for suicide attempts.

1.1.6.6 Psychomotor Activity

Psychomotor retardation is reported to be more common in adolescence than in children (Carlson & Kashini, 1988) but rates may not change across adulthood (Garvey & Schaffer , 1994; Wallace & Pfohl, 1995). In contrast, psychomotor agitation appears to be more common in children than adolescents (Ryan et al., 1987). Garvey and Schaffer (1994) found that psychomotor agitation was more common in both men and women between 40 and 80 years of age, but Wallace and Pfohl (1995) demonstrated that psychomotor agitation was more common with increased age in adult women, but not men. Overall, psychomotor retardation appears to present more commonly in adolescence from which time it appears stable across adulthood, whereas, psychomotor agitation appears to present more commonly in children but research regarding adulthood is mixed.

1.1.6.7 Vegetative Symptoms

Sleep

Insomnia and sleep difficulties appear to be common occurrences during MDD (Reynolds, 1999, Harvey, 2001). It has been estimated that in individuals experiencing an affective disorder, approximately 17 to 52% experience insomnia (see Harvey, 2001). In those experiencing major depression, insomnia is present in approximately 25% of individuals (Weissman, Greenwald, Nino-Murcia, & Dement, 1997).

Sleep During Childhood and Adolescent Depression

The literature on child and adolescent sleep changes in depression is mixed and sparse. Kaufman, Martin, King, and Charney (2001) found that only one of four studies found significant findings for rapid eye movement (REM) latency[1] differences in children with depression and no differences were found for delta sleep. It has also been asserted by some researchers that adolescents with depression show reduced REM latency (Kaufman, et al., 2001) and prolonged sleep latency[2], but no changes in delta sleep[3] or sleep continuity (Dahl, et al., 1996; Dahl, 1996). This is partially supported by a review (Benca, Obermeyer, Thisted, & Gillin, 1992) that revealed that the sleep of individuals with an affective disorder with mean ages less than 20 years was indistinguishable from that of controls, except for prolonged sleep latency. Others show different results, some have asserted that insomnia

[1] REM latency is the time taken to begin REM sleep.
[2] Sleep latency is the time taken to get to sleep.
[3] Delta sleep is the total slow wave sleep, it includes stages three and four of sleep.

occurs consistently across all ages (Carlson & Kashini, 1988). These findings of limited sleep disturbance associated with MDE during childhood and adolescent depression contrast those of adults where sleep disturbances are consistently found (see below).

Vegetative Differences Between Early- and Late-Onset Depression

Studies have also investigated differences in vegetative symptoms between early- and late-onset depression. Angold, Weissman, Wickramaratne, and Prusoff's (1991) community study of depressive symptomatology in individuals aged 6 to 23 years illustrated that from various groups of symptoms, vegetative symptoms (sleep and weight changes) were the most strongly affected by age. Weissman, et al. (1987) also found some small differences in vegetative symptom profiles between early (< 15 years) and later onset depression. The latter group more often reported weight loss and insomnia, and less often reported weight gain.

Adult Findings

In adults, sleep EEG abnormalities may be present in 40 to 60% of outpatients, and up to 90% of inpatients with a MDE. Polysomnographic findings conclude that sleep continuity disturbances (prolonged sleep latency, increased intermittent wakefulness, and early morning awakening), reduced non-rapid eye movement (NREM) slow-wave sleep, decreased REM latency, increased phasic REM latency[1], and increased duration of REM sleep early in the night, are the most frequently associated sleep difficulties with MDE (Reynolds & Kupfer, 1987; Van Moffaert, 1994; APA, 2000; Kaufman, et al., 2001). In support of these findings, Izikowski (1994) commented that depression is associated with an increased time to get to sleep, reduced sleep efficiency[2], early morning waking, reduced slow-wave sleep, reduced REM latency, and increased REM. Saletu-Zyhlarz, et al. (2002) studied middle-aged and older adults with depression (35-75 years) and found that their sample presented with decreased sleep efficiency, less total sleep time, less stage 2 sleep, increased wakefulness during the total sleep period, more early morning awakening, and greater sleep latency to stages 1, 2, 3. Regarding developmental changes, early and middle insomnia do not appear to vary across adulthood (Garvey & Schaffer, 1994; Wallace & Pfohl, 1995), but late insomnia[3] appears to be more common in those 40 to 80 years of age (Garvey & Schaffer, 1994). Overall, these studies from child, adolescent and adult samples suggest that the pattern of

[1] Phasic REM latency is the period before the onset of behaviours that occur periodically during REM sleep.
[2] Sleep efficiency is the ratio of total sleep time to total recording time multiplied by 100.
[3] Late insomnia is awakening before one's usual waking time and being unable to return to sleep.

adult sleep abnormalities in those with depression occurs less frequently or is only partially expressed in adolescent depression.

Normal Development of Sleep

Dahl et al (1992) has described the normal developmental changes in sleep. Typical children have very deep and highly efficient sleep, and are difficult to arouse from sleep and sleep for long periods of time. During adolescence sleep decreases 40% in duration and in the intensity of slow-wave sleep, a 30 to 40% decrement in REM latency occurs, and individuals experience greater sleepiness during the day. Adults, however, display a continued and steady decrease in slow-wave sleep, diminished sleep efficiency, decreased threshold of arousal from sleep and reduced REM latency. Knowles and MacLean (1990) suggested that patients with MDD appear to manifest these age-related changes in REM sleep, but at an accelerated rate. However, Benca, et al. (1992) found this theory doubtful, based on their findings that age-by-depression interactions were absent. Benca, et al., suggested that age may enhance the effects of depression on sleep where differences between those with depression and controls on sleep efficiency increased with age, but the differences in the length of the first REM period decreased with age. However, these trends were unable to be predicted by their relationships to either depression or age.

Hypersomnia

Hypersomnia has been found to occur in 15 to 26% of adult patients with MDD. (Reynolds & Kupfer, 1987; Billiard, Dolenc, Aldaz, Ondze, & Besset, 1994). Kovacs (1996) states that the most consistent empirical evidence regarding developmental changes in depression concerns hypersomnia. It seems that children present with less hypersomnia than adolescents, the rate then increases in adulthood and again drops in the elderly (Ryan et al., 1987; Garvey & Schaffer, 1994; Kovacs, 1996).

Appetite and Weight

Some research claims that weight *loss* associated with depression is more common in adolescence than in children (Goodyer & Cooper, 1993). Systematic differences in weight loss have not been found during adulthood (Wallace & Pfohl, 1995). Garvey and Schaffer (1994) demonstrated that appetite and weight *gain* were more common in those 40 to 80 years, but, as found by Wallace and Pfohl, appetite and weight loss did not change across adulthood.

1.1.6.8 Dysfunctional Cognitions

Beck's cognitive model (Beck, Rush, Shaw, & Emery, 1979) has significantly influenced the study of depression. His theory posits that the underlying assumptions held by individuals with depression play a significant role in the etiology and maintenance of depression. Consistent with Beck's theory, research has demonstrated that individuals with depression display higher mean scores on the Dysfunctional Attitudes Scale (DAS; Nelson, Stern, & Cicchetti, 1992), that depression severity is associated with the DAS (Norman, Miller, & Dow, 1988, Nelson, et al., 1992), and that individuals who report higher levels of dysfunctional attitudes and greater numbers of negative life events are more likely to develop depressive dysphoria (Klocek, Oliver, & Ross, 1997). However, there has been little empirical support for the hypothesised relationship between dysfunctional attitudes and the initial onset of depression (Ilardi, Craighead, & Evans, 1997) and some investigators found that the DAS scores, although correlated with depressive symptomatology, were not specific to depression (Silverman, Silverman, & Eardley, 1984; Hollon, Kendall, & Lumry, 1986; Hill, Oei, & Hill, 1989).

Previous research (Hamilton & Abramson, 1983; Norman, Miller, & Klee, 1983) has suggested that when acutely symptomatic, only 40 to 55% of individuals with depression have elevated scores on measures of dysfunctional cognitions. Consistent with this, Norman, et al. (1988) suggested that only a subgroup of inpatients with depression manifest elevated levels of dysfunctional cognitions. The subgroup included individuals who were younger at admission and at age of depression onset, those who had a significantly shorter duration of current depressive episode and tended to have a greater number of previous episodes, and those with poorer social functioning and a greater severity of depression. Again consistent with Beck's theory (Beck, et al., 1979) that dysfunctional attitudes are learned early in life, Norman, et al. suggest that the presence of elevated levels of dysfunctional cognitions are associated with a greater risk for depression at a younger age. However, others (Luty, Joyce, Mulder, Sullivan, & McKenzie, 1999) have shown, through step-wise regression, that 'duration of depression' accounted for the correlation of younger age and early age of onset with the DAS in a clinical sample.

These findings have noteworthy implications. Although dysfunctional cognitions may or may not be etiologically linked to depression, they are a key component in cognitive behavioural therapy (CBT; Beck, et al., 1979) Evidence suggesting that younger individuals

manifest greater levels of dysfunctional cognition's may provide some support for the use of CBT with younger individuals. If dysfunctional cognitions are a common risk factor for young people then a specific area of preventative work may have been identified. Yet, according to Luty, et al. (1999) age may not be a useful indicator.

1.1.6.9 Psychological, Social and Occupational Functioning

The psychosocial consequences of adults with depression at a two-year follow-up have been reported to include deficits in annual income, declines in job status, lower level of education, poor relationship satisfaction, lower likelihood of marriage, and if married, greater likelihood of divorce or separation, and deficits in social and leisure activities even among those no longer experiencing clinical symptoms (Coryell, et al., 1993). However, it has been demonstrated that while individuals experience depressive symptomatology they tend to over-report poor social adjustment (Morgodo, Smith, Lecrubier, & Widlocher, 1991). Early-onset depression and adolescent depression has also been associated with significant and persistent functional impairment (Puig-Antich, et al., 1993; Fombonne, Wostear, Cooper, Harrington, & Rutter, 2001). Research shows impairments in academic performance, truancy, or dropping out (Speier, Sherak, Hirsch, & Cantwell, 1995), family, marital, and social relationships, but not occupational and overall adjustment in adolescents (Garber, Kriss, Koch, & Lindholm, 1988). A study investigating the clinical outcomes of adolescent-onset depression into adulthood (10-15 year follow-up) also found a significant impairment in work, social and family life when compared to those without a psychiatric illness (Weissman, et al., 1999). The individuals presented with a lower educational achievement, lower social class, and more time out of work due to psychopathology. In high-school students, those who reported depressive symptoms also perceived less emotional support and warmth from friends and family members (Lewinsohn, et al., 1994; Greenberger, Chen, Tally, & Dong, 2000) and reported higher levels of hostility with close friends, and less reciprocal friendship relations (Windle, 1994). Childhood depression has been associated with impairments in academic performance, teacher-child relationships, and peer relationships (Puig-Antich, et al., 1985a; Puig-Antich, et al., 1985b). Overall, significant functional impairment associated with depression, across a variety of domains, is present at all ages. However, Luty, Joyce, Mulder, and McKenzie (2002) found that SAS total scores, friction scores and interpersonal behaviour scores declined with age 18 to 64 year old individuals, suggesting that social impairment associated with depression declines with age.

1.1.6.10 Summary

The research reviewed above suggested that the expression of some symptomatology may change with age. The only consistently reported symptom that appeared to increase with age across adulthood was late insomnia. Research regarding developmental changes in other symptoms including psychic anxiety, irritability, suicidal ideation, weight and appetite changes, and dysfunctional cognitions was mixed. Symptoms that appeared to be uninfluenced by age across adulthood include depressed mood, anhedonia, somatic anxiety, psychosis, suicidal attempts, psychomotor retardation, insomnia and functional impairment. By extending the literature review to adolescence, further developmental differences were seen. Symptoms that appeared to be more common in adolescents than children included anhedonia, irritability, psychomotor retardation and weight changes. The only consistent symptom that appeared to be more common in adolescents than adults was suicide attempts. Symptoms that appeared to be more common in adults than adolescents included delusions and sleep disturbances except prolonged sleep latency and hypersomnia. Research regarding other symptoms was either mixed, or was not found.

1.1.6.11 Methodological and General Considerations

Again, methodological differences may have contributed to the mixed findings in symptomatology. For example, Garvey and Schaffer (1994) assessed participants with the Affective Disorders and Schizophrenia (SADS; see Garvey & Schaffer, 1994), whereas, Wallace and Pfohl (1995) assessed individuals with the HDRS-24 (Hamilton, 1960, cited in Wallace and Pfohl, 1995). In addition, Garvey and Schaffer split their sample into those under and over the age of 40, whereas, Wallace and Pfohl studied one sample aged 18 to 81. Some sources of information were review articles (e.g., Kovacs, 1996; Kaufman, et al., 2001). Some studies assessed solely adulthood (e.g., Wallace & Pfohl), whereas, others studied preschoolers, prepubertal children, adolescents, and adults (e.g., Carlson & Kashini, 1988). Finally, some studies used community samples (e.g., Angold, et al., 1991), whereas others used clinical samples (e.g., Garvey & Schaffer, 1994).

1.1.7 Comorbidity

Comorbidity generally refers to the manifestation of two or more disorders whose co-occurrence is greater than what would be expected by chance alone (Mash & Dozois, 1996). Hence, in this thesis it refers to other disorders that co-occur with depression. The DSM-IV-TR (APA, 2000) utilises a multiaxial system to assess and classify psychiatric disorders. Axis

I includes clinical disorders and other conditions that may be a focus of clinical attention, Axis II includes personality disorders and mental retardation, Axis III includes general medical conditions, Axis IV includes psychosocial and environmental problems, and Axis V includes a global assessment of functioning. Disorders from Axes I and II will be mentioned below to allow for a discussion of comorbidity with depression.

1.1.7.1 Axis I: Clinical Disorders

A review by Angold and Costello (1993) reported a high rate of comorbidity in children and adolescents with MDD or dysthymia. Comorbidity with conduct disorder (CD), oppositional defiant disorder (ODD), anxiety disorders, and attention deficit disorder was common. Substance abuse, alcohol abuse, (Birmaher, et al., 1996) and eating disorders (APA, 2000) have also been identified as common comorbidities of depression in adolescents. Adult depression has been reported to be comorbid with a variety of psychiatric diagnoses, including schizophrenia, anxiety disorders (29%; Schatzberg, Samson, Rothschild, Bond, & Regier, 1998), personality disorders, alcoholism (Angold, 1993), and chronic pain (Fishbain, Cutler, Rosomoff, & Rosomoff, 1997).

Kovacs (1996) concluded, from a review, that the rate of comorbidity with any axis I or II psychiatric disorder among children and youths (80-95%) is similar to, or, only slightly higher than, comorbidity rates in adults (60-90%). Kovacs study reported comorbidity rates of approximately 33% in children and adolescents and 40 to 50% in adults for anxiety disorders. Twenty-five percent of adults experienced a substance use disorder. The rate for substance use disorder was low in the youth, however, the rate for conduct disorder which is sometimes considered an equivalent in youth was 15%.

Axis I Comorbidity in Early- Versus Late-Onset MDD

Research has shown differences in comorbidity rates in those with early- and late-onset depression. Alpert, et al. (1999) demonstrated that social and simple phobias and alcohol abuse and/or dependence were significantly more prevalent in those with childhood-onset MDD than those with adult-onset. Alcohol abuse and/or dependence was significantly more prevalent among adolescent-onset than adult-onset MDD. Panic, generalized anxiety, obsessive-compulsive, and somatoform disorders were equally distributed across all groups.

Summary

Overall, although it is difficult to compare age groups due to different diagnoses, it appears that within a similar comorbidity rate, children, adolescents and adults present with different patterns of comorbidity, which may be mediated by age at onset of the depressive disorder.

1.1.7.2 Axis II: Personality Disorders

The comorbidity rate of personality disorders (PD) in depressed adult samples ranges from 30 to 70% (Farmer & Nelson-Gray, 1990). Ampollini, et al. (1999) found higher rates of obsessive-compulsive personality disorders in patients with depression when compared to individuals without a psychiatric condition and individuals with panic disorder. In addition, Curruble, Ginestet, and Guelfi (1996) found higher rates of cluster B[1] PD's in individuals with MDD, while others have not (Ampollini, et al., 1999). Cluster C[2] PD's have been show to be greater in those with MDD than those without a psychiatric disorder, yet this was also true for panic disorder (Ampollini, et al., 1999). Hence, cluster C comorbidity appears to be non-specific to depression.

Axis II Comorbidity in Early- Versus Late-Onset MDD

Research has shown higher rates of various personality disorders in early-onset depression when compared to late-onset depression. Early-onset (< 18 years) depression has been associated with a higher prevalence of avoidant, histrionic, narcissistic, and borderline personality disorders (Fava, et al, 1996).

Causality

There is some evidence suggesting that axis II pathology predisposes individuals to experience depressive episodes. Ilardi and Craighead (1999) reported that 29% of the variance in the DAS was accounted for by axis II pathology in formerly depressed inpatients, suggesting a trait-like characteristic placing them at a greater vulnerability to experience subsequent depressive relapse episodes. However, the influence of age was not known in these findings. Other research has also shown that PD's may place individuals at risk for depression indirectly by increasing episodic stress and interpersonal chronic stress (Daley,

[1] Cluster B includes the Antisocial, Borderline, Histrionic and Narcissistic PD's. Individuals with these disorders often appear dramatic, emotional, or erratic. [2] Cluster C includes the Avoidant, Dependent, and Obsessive-Compulsive PD's. Individuals with these disorders often appear anxious or fearful.

Hammen, Davila, & Burge, 1998). Whereas another study has shown that gender mediates the relationship between PD's and axis I disorders where internalising and externalising symptoms predicted cluster B symptoms in girls and cluster B symptoms predicted externalising symptoms in boys. In addition to PD's placing individuals at risk for depression, depression may place individuals at risk for developing PD's (Kasen, et al., 2001). For example, depression may disrupt socialisation processes and personal development during critical periods in adolescence or early adulthood (Kovacs & Goldston, 1991) which may later place individuals at risk of developing a PD.

1.1.8 Risk Factors

A number of risk factors for depression have been identified including genetic factors, lack of parental care, gender, certain personality traits, and childhood sexual abuse (see Joyce, 2000). Research has demonstrated differences in risk factors for MDD between early- and late-onset MDD (Jaffee, et al., 2002). Therefore, the occurrence of identified risk factors may differ as a function of age. The occurrence of risk factors including personality, familial history of affective disorders and alcohol dependence, childhood sexual abuse and parental bonding during adolescence and adulthood will now be discussed.

1.1.8.1 Personality

A Psychobiological Model

As mentioned earlier, axis II PD's may place individuals at risk of developing depressive episodes. Other research has produced significant findings surrounding personality traits and depression. A psychobiological model of the structure and development of personality in terms of temperament and character has been developed by Cloninger, Svrakic, and Pryzbeck (1993). The four temperament dimensions are thought to be moderately heritable, moderately stable throughout life, and invariant despite sociocultural influences. The first temperament dimension is novelty seeking which is defined as a heritable bias in the activation of behaviours such as frequent exploratory activity in response to novelty, impulsive decision making, extravagance in approach to cues of reward, and quick loss of temper and active avoidance of frustration. Secondly, harm avoidance is a heritable bias in the inhibition of behaviours such as pessimistic worry in anticipation of future problems, passive avoidant behaviours such as fear of uncertainty and shyness of strangers, and rapid fatigability.

Reward dependence is a heritable bias in the continuation of ongoing behaviours and is manifest through sentimentality, social attachment, and dependence on approval of others. The forth dimension, persistence, is characterised by perseverance despite frustration and fatigue. The character dimensions are thought to mature in adulthood and influence personal and social effectiveness by insight learning about self-concepts such as the extent to which one identifies the self as an autonomous individual, an integral part of humanity, and an integral part of the universe as a whole. The first character dimension is self-directedness and it refers to self-determination or 'willpower'. Individuals high on self-directedness are usually described as responsible, resourceful and disciplined. Cooperativeness is related to agreeability and individuals high on cooperativeness are described as socially tolerant, empathetic, helpful and compassionate. Finally, self-transcendence refers generally to the identification with everything conceived as essential and consequential parts of a unified whole (Cloninger, Svrakic, & Pryzbeck (1993); Svrakic, Svrakic & Cloninger, 1996).

Personality Dimensions and MDD

Some research has shown that high harm avoidance levels characterise adults with MDD (Joyce, Mulder, Cloninger, 1994; Ampollini, et al., 1999) and more recently Ampollini, et al. found that reward dependence also characterises individuals with MDD. However, Hansenne, et al. (1999) reported that patients with depression exhibited higher harm avoidance and self-transcendence scores and lower self-directedness and cooperativeness scores when compared to individuals without a psychiatric condition. The authors found that harm avoidance, self-directedness and cooperativeness dimensions were related to the severity of depression as assessed by the Hamilton Depression Rating Scale (Hamilton, 1967). Meanwhile, Luty, et al. (1999) demonstrated the strongest predictors of dysfunctional attitudes (accounting for 45% of the variance) to be duration of depression, reward dependence, and self-directedness, but primarily self-directedness. Luty, et al., suggest that these findings may provide an alternative framework for understanding a 'core cognitive vulnerability' to depression and provide some insight into the high comorbidity of personality disorders in those with depression. No evidence is available regarding the influence of age on personality traits in those with depression.

Personality Dimensions and Normal Development

Cloninger, et al (1993), studied developmental differences in character in individuals from the general population. Results indicated that scores on the self-directedness and

cooperativeness subscales increased with age, but self-transcendence was not significantly associated with age. Regarding temperament, Svrakic et al. (1996) described temperament as moderately heritable, and moderately stable throughout life. Hence, some developmental differences may occur, but they have not been documented.

1.1.8.2 Familial History of Affective Disorders and Alcohol Dependence

Affective Familial History Differs Between Early- and Late-Onset MDD

Research has shown that the earlier the age at onset of depression (especially before the age of 20), the higher the familial loading and specificity of familial transmission (Weissman, et al., 1984; Neuman, Geller, Rice, & Todd, 1997; Lyons, et al., 1998; Klein, Lewinsohn, Seeley, & Rhohde, 2001). Weissman, et al. (1984) found that 24% of first degree relatives (FDR's) of early-onset (< 20 years) probands were diagnosed with MDD, compared to 18% of FDR's of probands with an onset between 20 and 29, 12% of FDR's of probands with an onset between 30 and 39, and 8% of FDR's of probands with an onset greater than 39 years. More recently a study has shown that risk factors differ between juvenile- (ages 11, 13, and 15) and adult-onset MDD (ages 18, 21, and 26) where family psychopathological characteristics posed a greater risk in juvenile-onset MDD (Weissman, 2002). In support of this, Wickramarantne and Weissman (1998) found that children of depressed parents were at a high risk for juvenile-onset depression due to the early parental age at onset of MDD. A surprising finding was presented by Kendler, Gardner, and Prescott (1999). This study contradicted at least six other family studies, it did not find age of onset or gender to be significantly related to a familial history of MDD. Instead, the best predictors of familial history of MDD included number of episodes, duration of longest episode, recurrent thoughts of death or suicide, and level of distress or impairment. Kendler, et al.'s study has been criticised by Weissman and Wickramaratne (2000) for poor methodology, hence replication of this study is necessary.

Affective Familial History in Pre-Pubescent-Onset MDD

Although much research supports the notion of greater familial loading of depression in early adult-onset depression, this finding does not appear to extend in a simple linear fashion down to pre-pubescent-onset cases (Harrington, et al., 1997). Family studies of child and adolescent probands with affective disorders have shown high rates of affective disturbance among FDR's (Goodyer, Cooper, Vize, & Ashby, 1993; Harrington, et al., 1993) which could reflect a greater genetic risk. However, to complicate the issue, others have

found lower heritability in pre-pubescent-onset MDD (Thapar & McGuffin, 1996; Eley & Stevenson, 1999). Harrington, et al. (1997) reported that the familial loading of depression did not increase systematically with decreasing age. Specifically, FDR's of those with pre-pubescent-onset depression tended to have lower rates of depression than the relatives of probands with adolescent-onset MDD. Overall, it appears that there is a greater familial loading of depression in early adult-onset depression but the genetic influence does not systematically increase with decreasing age over adolescence and childhood.

Familial History of Alcoholism in Those with MDD

In addition to the transmission of familial affective disorders, research has shown that the depressed FDR's of pre-pubescent depressed children have significantly greater lifetime rates of alcohol-use disorder than the relatives of post-pubescent depressed children (Harrington, et al., 1997). Puig-Antich, et al. (1989) found that pre-pubescent-onset MDD is associated with 'familial comorbidity' of depression and alcoholism. However, no other disorder other than depression was increased among FDR's of post-pubescent-onset depression (Harrington, et al., 1997). Therefore, the authors suggested that pre-pubescent-onset depression may be just as heritable as post-pubescent- or adult-onset depression, but with a different genotype (Harrington, et al., 1997). Rende, et al. (1997) studied adults and found the FDR's of early-onset (< 20 years) probands had increased rates of alcoholism, when compared to the FDR's of adult-onset probands, only if they also had MDD. Hence, alcohol abuse does not appear to place children at risk unless it is comorbid with depression and 'familial comorbidity' appears to extend up to approximately 20 years of age.

1.1.8.3 Childhood Sexual Abuse

Approximately 10 to 25% of females and 2 to 10% of males report exposure to childhood sexual abuse (CSA; Fergusson, Lynskey, & Horwood, 1996) and those who report CSA have higher rates of major depression, anxiety disorders, conduct disorder, substance use disorder, and suicidal behaviours after controlling for antecedent childhood factors (Fergusson, Horwood, & Lynskey, 1996). CSA has been established as a risk factor for adult major depression (Joyce, 2000). CSA has been shown to be a significant risk factor in both juvenile- (11-15 years) and adult-onset (18-26 years) depression in a community sample (Jaffee, et al., 2002). Therefore, it appears that it may consistently appear as a risk factor across adolescence and adulthood.

1.1.8.4 Parental Bonding

Several theories such as attachment theory and object relations theory, suggest that interactions with early caretakers provide a basis for negative information processing structures, however, there has been little empirical research to assess this association (Ingram & Ritter, 2000). It is thought by some researchers that internal working models of relationships are central to understanding depression because secure and disturbed patterns of caring relationships are internalised by children as mental representations. When these representations are impaired due to disturbed relationships they can create a vulnerability to later depression (Blatt & Homann, 1992). Lack of care[1] and overprotectiveness[2] are widely agreed to represent the critical bonding dimensions related to the development of psychopathology (Ingram, Overbey, & Fortier, 2001). It is these dimensions of care and overprotectiveness that are measured by the Parental Bonding Instrument (PBI; Parker, Tupling & Brown, 1979).

Research using the PBI suggests that individuals who experience disrupted parental bonding, particularly deficits in care, are at risk of developing depression (Gerlsma, Emmmelkamp, & Arrindell, 1990). However, when looking more specifically, the research is mixed. Some investigators found that only disrupted *maternal* care increased the risk of recent depression (Mackinnon, Henderson, & Andrews, 1993; see also Oakley-Browne, Joyce, Wells, Bushnell, & Hornblow, 1995) and others found only an association between *paternal* care and depression (see Oakley-Browne, et al., 1995). To complicate the issue further, some research has demonstrated that low care is linked with an increased chance of psychopathology in general, not specifically to depression (Parker, Hadzi-Pavolvic, Greenwald, & Weissman, 1995).

Parental Bonding and Age

The effect of age on the PBI was found to be non-significant suggesting that a change does not occur in the reporting of parental attitudes over time nor do individuals responses change as they become more distant from childhood (Parker, et al., 1979). However, this has not been researched in a sample of individuals with depression.

[1] Lack of care is characterised by "emotional coldness, indifference and neglect".
[2] Overprotectiveness is characterised by "control, overprotection, intrusion, excessive contact, infantilisation".

Parental Bonding and Gender

With regard to gender, research has shown that within a normal sample mothers were experienced as more caring and more overprotective than fathers, however, the gender of the respondent did not influence either parents score (Parker, et al., 1979). Parker (1983) reported that women with depression tended to report low caring and high overprotection ("affectionless control") in their mothers while men tended to report "affectionless control" in their fathers. Carter, Joyce, Mulder, Luty, and Sullivan (1999) stated that females with depression were not more vulnerable to adverse early parental care than males. Therefore, it appears that while gender may influence how an individual views their parents, it does not contribute to greater vulnerability.

Parental Bonding and Cognitive Deficits

Poor parental bonding may place individuals at risk for depression directly or indirectly. For example, irrelevant of gender, Parker (1979) reported that lower maternal care was associated with depressive symptoms and with the kind of cognitive deficits seen in depression. Ingram, et al.'s (2001) findings support this. These authors demonstrated that lower maternal care was associated with deficits in positive cognition and excesses in negative cognition. In addition, Ingram and Ritter (2000) found that formerly depressed individuals with lower levels of maternal care diverted their attention toward negative stimuli when they were in a sad mood more than those never depressed and those with higher levels of maternal care. Perhaps, the increased risk that arises from poor parental bonding could be partially explained by increased levels of dysfunctional thinking. Alternatively parental bonding may place individuals at risk indirectly via personality or life events. Joyce (2000) explained that while parental bonding does not significantly directly predict liability to depression in women, variables such as parental warmth impact indirectly by influencing neuroticism, a history of past depressive episodes, recent difficulties, and lifetime traumas. Hence, there are a number of variables through which poor parental bonding may place individuals at risk for MDD indirectly.

1.2 Rationale for this Thesis

This literature review has shown both consistent and inconsistent findings regarding the effect of age on depressive symptomatology, comorbidity, and the occurrence of identified risk factors. The majority of the research investigating developmental differences in symptoms of MDD was inconclusive due to mixed findings. Symptoms that did appear to

consistently change in occurrence with age included early insomnia, late insomnia, hypersomnia, anhedonia, irritability, psychomotor retardation, appetite and weight gain, suicide attempts, and delusions. However, the specific differences between early and middle adulthood were not shown because previous studies often correlated the occurrence of symptoms across adulthood which included older adults. The rates of axis I comorbidity did not appear to change significantly over age except that alcohol abuse and/or dependence, and social and simple phobias were more common in early-onset MDD. Axis II comorbidity was significantly different between early- and late-onset MDD. Younger individuals appeared to present with more PD's, especially avoidant, histrionic, narcissistic, and borderline PD. However, studies of early- and late- onset depression compare adolescents with adults, therefore, changes across adulthood remain unknown. With respect to risk factors, the relationship between dimensions of personality and age in those with depression had not been studied. The majority of the research supported the notion that early-onset MDD had greater familial loading of affective disorders. Familial loading of alcoholism in those with MDD also appeared to be greater in those with early-onset MDD. Childhood sexual abuse did not appear to differ as a risk factor between early- and late-onset MDD. Finally, the occurrence of parental bonding as a risk factor did not appear to be associated with age. Again, research on the occurrence of risk factors across early and middle adulthood is lacking. Overall, it appears that age is influential and requires further investigation particularly with reference to changes during both pre- and post-pubescent adolescent onset depression and across early and middle adulthood depression because adulthood is generally combined into one bracket, or correlations across all of adulthood are used. Thus it is important to investigate differences between adults because differences in symptomotology and comorbidity could aid in the psychiatric assessment of depression in adults. In addition, differences in risk factors would have significant implications for the clinical understanding of the etiology of depression, and in turn, treatment.

1.2.1 Differential Responses to Medication Across Age

A further rationale for this thesis comes from pharmacological research. Joyce, et al. (1997) found that fluoxetine was more effective than nortriptyline when treating adults with depression. However, further analyses revealed that the greater efficacy of fluoxetine in those 25 years and younger explained the significant difference between the medications. Hence, the authors concluded that selective serotonin reuptake inhibitors (SSRI) were more effective for younger individuals (< 25 years) than tricyclic antidepressants (TCA), and SSRI's and

TCA's were both effective for individuals older than 25. This finding extends previous literature with children and adolescents that showed poor responses to TCA's (Geller, Cooper, Graham, Fetner, Marsteller and Wells, 1992; Kutcher, et al., 1994; Wagner & Ambrosini, 2001) and positive results from SSRI's (Boulos, Kutcher, Gardner, & Young, 1992; Colle, Belair, DiFeo, Weiss, & LaRoache, 1994; Emslie, et al., 1997). These findings suggest that younger adults respond to similar pharmacological treatment as children and adolescents, and different pharmacological treatment than those above 25 years of age. Obviously, age is an important variable in the pharmacological treatment of MDD. This raises the question of, the age at which adolescence may be said to end with regard to depression. Furthermore, perhaps age is influential in other aspects of MDD.

1.2.2 A Theory of 'Emerging Adulthood'

From a sociological perspective, there is controversy about when adolescence ends and early adulthood begins. Arnett (2000) argues that the late teens and early twenties is no longer a time of entering and settling into long-term adults roles because contemporary Western cultures allow young people a prolonged period of independent role exploration, characterised by frequent change and exploration. Arnett proposes that 'emerging adulthood' occurs from 18 to 25 which is neither adolescence or young adulthood. He describes 'emerging adulthood' as being a time of "relative independence from social roles and from normative expectations" and a time to explore love, work, and worldviews.

Within contemporary Western cultures, where independence, individualism, and self-expression are usually highly valued, the entrance into adulthood is typically defined by residential and financial independence, cognitive self-sufficiency, emotional self-reliance, and behavioural self-control. This is in contrast to socially defined entrances to adult roles in non-Western cultures such as the social event of marriage, or its equivalent, where obedience and conformity are often highly valued and adulthood is marked communally, not individually (Arnett & Taber, 1994). Hence, while university students are often used in research representing adults, they may be distinctly different from adults and possibly more like adolescents in some ways, as suggested by the pharmacological findings and sociological perspective above. This has substantial implications for the study of adulthood. Currently findings are generalised from young adults to represent all adults. Therefore, studies investigating early, middle, and late adulthood are necessary.

1.2.3 Hypotheses and Aims

This thesis will investigate developmental differences in depression by investigating trends in the expression of depression, comorbidity and the occurrence of identified risk factors for depression during young and middle adulthood. It is expected that, based on previous research, differences in symptomatology between young adults and middle adults will be significant. Specifically, late insomnia will be more common in middle-aged adults, whereas, irritability, suicide attempts, early insomnia, appetite and weight gains, and hypersomnia, will be more common in young adults. However, it is expected that depressed mood, anhedonia, psychosis, appetite and weight loss, psychomotor retardation, middle insomnia and functional impairment will occur at similar rates during early and middle adulthood. Regarding other symptomatology, the literature is sparse or inconsistent hindering any further predictions. It is expected that young and middle-aged adults will have similar profiles of axis I comorbidity, except younger individuals will show greater rates of alcohol and/or drug dependence. Regarding axis II comorbidity, younger adults are expected to present with greater rates of avoidant, histrionic, narcissistic and borderline PD's. Childhood sexual abuse and parental bonding will be consistent risk factors in both young and middle adults. However, the rate of familial affective and alcohol dependence disorders will decrease with age. Differences on personality dimensions are expected to mimic that of normal development. That is, scores of self-directedness and cooperativeness will be higher in middle-aged adults and the other dimensions of personality will show similar scores across early and middle adulthood.

CHAPTER 2

METHOD

2.1 STUDIES

This thesis analysed data previously collected by the Department of Psychological Medicine, Christchurch School of Medicine. Data came from three large sequential clinical studies investigating depression.

2.1.1 Study One: Predictors of Response to Clomipramine or Desipramine

This study was a six-week, double-blind, randomised clinical trial of pharmacological treatment for depression. The aim of this study was to investigate predictors of response to clomipramine or desipramine in depressed outpatients.

Participants were between the ages of 18 to 64 (N=112), with a principal diagnosis of a DSM-III-R (APA, 1987) MDE when entering the study. They had been drug free for a minimum of two weeks (except for the oral contraceptive pill or an occasional hypnotic), and were not breast-feeding. Participants were excluded from the study if they had a serious physical illness which may have interfered with assessment or treatment, a history of schizophrenia, schizoaffective disorder or mania, a current alcohol or drug dependence of moderate or greater severity which was deemed to be the principal diagnosis, or had been treated with the antidepressants used in this trial during the previous 12 months.

2.1.2 Study Two: Predictors of Response to Fluoxetine or Nortriptyline

This was a five-year prospective study, but with initial randomisation to fluoxetine or nortriptyline. The aim of the study was to investigate predictors of response to fluoxetine or nortriptyline in depressed outpatients (N=195).
Participants met the same criteria as those in study one.

2.1.3 Study Three: Predictors of Response to Interpersonal Psychotherapy and Cognitive Behaviour Therapy.

This study was a 13-week, randomised clinical trial of psychotherapy for depression, and is still in progress. The aim of this study was to examine predictors of response to Interpersonal Psychotherapy (IPT) and Cognitive Behaviour Therapy (CBT) for depression.

Participants were over the age of 18 (N=155), had a principal diagnosis of a DSM-III-R (APA, 1987) MDE when entering the study, and were drug free for a minimum of two weeks or five drug half-lives (except the oral contraceptive pill or an occasional hypnotic). Participants were excluded from the study if they had a serious illness which may have interfered with assessment or treatment, had severe or psychotic depression, a severe antisocial personality disorder, a history of schizophrenia, or bipolar I disorder, a current alcohol or drug dependence (excluding cannabis) of moderate or greater severity which was deemed to be the principal diagnosis, were receiving any other therapy for depression, or had failed to respond to an adequate trial of CBT or IPT in the past year.

2.2 PROCEDURE

All participants for the three studies were referred from a variety of sources including general health practitioners, psychiatric emergency services, other mental health services and self-referral. When referred to the studies, participants were screened by a psychiatric nurse who assessed their eligibility. Following this procedure, those potentially eligible underwent a psychiatric assessment to confirm their eligibility based on the inclusion and exclusion criteria stated above. Following the giving of written informed consent and prior to treatment, a detailed biological and clinical assessment and a battery of self-report measures were completed. The protocols for all three studies were approved by the Canterbury Ethics Committee. Throughout treatment and during a follow-up period, participants were continually assessed.

2.3 CURRENT THESIS

2.3.1 Participants

Participants in this thesis were or are currently enrolled in one of the three studies described above. This thesis utilised data from 447 participants aged 18 to 54 years: 110 from study one, 188 from study two, and 149 from study three. These were all the participants included in the above studies within the age range of 18 to 54 years. The upper limit of 54 was used because, as mentioned, this thesis aims to compare young adults with adults in middle adulthood, therefore excluding older adults.

Design similarities and sample characteristics (see Table 1) enabled some of the data from all three studies to be combined to provide a larger sample size for analyses. Of the

entire sample (N=447), 62% were women and 38% men; 48% were never married, 34% were currently married or had been living with their partner for one or more years, and 19% were separated, divorced or widowed; 94% were of European decent, 3% Maori, and 4% were of Pacific Island, Asian or other decent[1].

Table 1

Demographic Data from the Three Studies

		Study one *N*=110		Study two *N*=188		Study three *N*=149	
		M	*SD*	*M*	*SD*	*M*	*SD*
Age (years)		31.03	9.89	30.08	9.90	34.24	9.35
Depression severity	HDRS 17-total	21.09	5.20	19.93	4.32	16.52	5.02
		%	*n*	%	*n*	%	*n*
Gender	Women	54.54	(60)	57.97	(109)	71.81	(107)
Marital status	Single	49.09	(54)	51.60	(97)	41.61	(62)
	Married/living with partner	28.18	(31)	30.32	(57)	42.28	(63)
	Separated/divorced/widowed	22.73	(25)	18.09	(34)	16.11	(24)
Ethnicity	Pakeha	92.73	(102)	95.74	(180)	91.28	(136)
	Maori	2.73	(3)	2.66	(5)	2.68	(4)
	Other	4.55	(5)	1.60	(3)	6.04	(9)
Principle diagnosis	MDD	91.82	(101)	90.43	(170)	95.30	(142)
	Bipolar II	8.18	(9)	9.57	(18)	4.70	(7)

2.3.2 Procedure

All assessment data reported in this thesis are baseline data. Thus, the data was obtained after consenting, and before treatment commenced. However, the SCID-II and family history interviews were administered 6 weeks into treatment to minimise the state effects of low mood on PD diagnosis and to allow a period of education about depression anticipating that participants may more readily recognise affective disorders in their relatives. The structured clinical interviews and clinician rated scales were administered by a psychiatrist or clinical psychologist and other measures, including self-report measures, were administered by a psychiatric nurse.

[1] Some of these percentages do not have a sum of 100 due to rounding errors.

2.4 MEASURES

Demographic data was collected by interview from each participant. The clinician rated depressive criteria were assessed with the Structured Clinical Interview for the DSM-III-R- Patient Version (SCID-P), and the Global Assessment of Functioning Scale (GAF). The clinician rated depressive symptoms were assessed with the Hamilton Depression Rating Scale (HDRS), and the Montgomery and Asberg Depression Rating Scale (MADRS). The self-report depressive symptoms and functioning were assessed with the Hopkins Symptoms Checklist – Revised (SCL-90-R), Dysfunctional Attitudes Scale (DAS), and Social Adjustment Scale- modified (SAS). Axis I comorbidity was assessed with the SCID-P, and Axis II comorbidity was assessed with the Structured Clinical Interview for DSM-III-R and DSM-IV, Axis II Personality Disorders (SCID-II). Risk factors were assessed with the Tri-dimensional Personality Questionnaire (TPQ), Temperament and Character Inventory (TCI), childhood sexual abuse interview, family psychiatric history interview, and the Parental Bonding Inventory (PBI). The structured clinical interviews, clinician rated scales and self-report measures used are described below.

2.4.1 Clinician Rated Depressive Criteria

2.4.1.1 Structured Clinical Interview for the DSM-III-R-Patient Version (SCID-P) (Spitzer, Williams, Gibbon, & First, 1992)

The SCID-P is a clinician administered, structured diagnostic interview which assesses current and lifetime axis I psychopathology based on DSM-III-R criteria. Modifications were made to the original SCID-P in study two and three to include DSM-IV (APA, 1994) melancholia and atypical features of depression and information about self-mutilation and suicidal behaviour. The alterations relevant to this thesis include:

1) Questions about current and past major depressive symptoms had extra subdivisions for:
    i) weight loss or gain
    ii) insomnia or hypersomnia
    iii) psychomotor agitation or retardation
    iv) recurrent thoughts of death, suicidal ideation, suicide plan, non-violent suicide attempt, and violent suicide attempt.
2) Questions regarding self-mutilation were included.
3) Greater detail regarding suicidal behaviour was included.

2.4.1.2 Global Assessment of Functioning Scale (GAF) (APA, 1994)

The GAF is axis V of the DSM-IV and is a clinician rated assessment of an individual's psychological, social and occupational functioning on a hypothetical continuum of mental health-illness ranging from 0-90. It is a commonly used method for assessing the global functioning of individuals suffering from a psychiatric illness. Clinicians consider both symptomatology and functional impairment and rate within one of 10 intervals. Following the rating, the individual's impairment in functioning is qualitatively described as mild (61-70), moderate (51-60), and severe (41-50). The GAF has produced satisfactory reliability and validity, and specifically it has been associated with individual's current support needs (Jones, Thornicroft, Coffey & Dunn, 1995).

2.4.2 Clinician Rated Depressive Symptoms

2.4.2.1 Hamilton Depression Rating Scale (HDRS) (Hamilton, 1967)

The HDRS is an observer rating scale designed to measure the severity of depression in individuals already diagnosed as suffering from a depressive disorder. The scale emphasises somatic, vegetative symptoms and a few items assess cognitive symptoms including guilt. The rating scale enquires about the previous seven days. In study one, the 17-item form was used. In studies two and three, the expanded 27-item form was used. Items are measured on a 3-point or 5-point scale resulting in a total score ranging from 0 to 52 on the 17-item and 0 to 82 on the 27-item.

Inter-rater reliability (*k*) ranged from .87-.98 in several studies (see Akdemir, et al., 2001), and item-total correlations (r) ranged from .21-.78 in two studies described by McNamara (1985). Test-re-test reliability over five days was .85, with a Cronbach alpha coefficient of .75, and it had a split-half reliability coefficient of .76 (Akdemir, et al., 2001).This scale has been well-validated in many studies (Santor & Coyne, 2001; Akdemir, et al., 2001).

2.4.2.2 Montgomery and Asberg Depression Rating Scale (MADRS)

(Montgomery & Asberg, 1979)

The MADRS is a 10-item (7-point scale) clinician rated depression severity scale. The scale was designed to provide a sensitive measure of depression severity, particularly for change during treatment. The items include apparent and reported sadness, inner tension, reduced sleep and appetite, concentration difficulties, lassitude, inability to feel, pessimistic

and suicidal thoughts. Hence, it has fewer somatic items than the HDRS. An advantage of this scale is that it has clear comprehensive definitions of its items and the scale steps. Research suggests good inter-rater reliability and sensitivity to change over time (Korner et al., 1990; Montgomery & Asberg, 1979).

2.4.3 Self-Report Depressive Symptoms and Functioning

2.4.3.1 Hopkins Symptoms Checklist – Revised (SCL-90-R) (Derogatis, 1983)

The SCL-90-R is a 90-item self-report questionnaire measuring current psychiatric symptomatology. Individuals complete the questionnaire by rating a five-point Likert scale regarding how frequently they have experienced symptoms over the previous seven days. There are nine subscales: somatisation, obsessive-compulsive difficulties, interpersonal sensitivity, depression, anxiety, anger-hostility, phobic anxiety, paranoid ideation and psychoticism. Research shows reliability coefficients have ranged from .84-.87 (Derogatis, Lipman, Rickels, Uhlenhuth, & Covi, 1974) and the validity has been demonstrated in a variety of studies with psychiatric and normal samples (Bech, et al., 1992; Deragatis & Cleary, 1977).

2.4.3.2 Dysfunctional Attitudes Scale (DAS) (Weissman & Beck, 1978)

The DAS is a 40-item self-report questionnaire. It is a measure beliefs held by individuals experiencing depression. The items such as "People will probably think less of me if I make a mistake" are rated on a seven-point degree-of-belief scale (1= not at all, 7= totally). Total scores were obtained by summing the 40 items resulting in scores ranging from 40-280 where higher scores indicate a greater endorsement of depressive set of attitudes or beliefs. Test-re-test reliability has been reported as .89 and the internal consistency as .89 (Carro, Bernal & Vea, 1998). The DAS has been shown to be a valid measure of dysfunctional cognitions in individuals with depression (Nelson, et al., 1992) and it has been shown to differentiate between depressed and non-depressed individuals (Carro, et al., 1998). However, others found that while the DAS correlates with depressive symptomatology, it may not be specific to depression (Hollon, et al., 1986; Hill, et al., 1989).

2.4.3.3 Social Adjustment Scale- Modified (SAS) (Cooper, Osborn, Gath, & Fegetter, 1982)

The original SAS was a 45-item self-report questionnaire assessing social functioning over the past two weeks (Weissman & Bothwell, 1976). The modified SAS contains eleven

subscales investigating social functioning over a variety of settings such as at work, with parents, or with their spouse. The subscales are: work outside the home, housework, social and leisure activities, extended family, marital, parental, family unit, role performance, interpersonal relationships, friction, feelings and satisfaction in work, and social and leisure activities. Higher scores on the subscales or the total adjustment score indicate lower levels of social functioning. Changes in the modified version include making the wording more appropriate to the United Kingdom, and use of the same scale throughout the questionnaire. Research shows significant correlations between clinical and self-report ranging from .40 to .72, with an overall agreement of .72 (Weissman & Bothwell, 1976).

2.4.4 Axis I and Axis II Comorbidity

2.4.4.1 Structured Clinical Interview for the DSM-III-R-Patient Version (SCID-P) (Spitzer et al., 1992)

As described above.

2.4.4.2 Structured Clinical Interview for DSM-III-R and DSM-IV, Axis II Personality Disorders (SCID-II) (First, Spitzer, Williams, & Gibbon, 1995; First, Gibbon, Spitzer, & Williams, 1997)

The SCID-II is a structured diagnostic interview which assesses axis-II personality disorders. The DSM-III-R version of the SCID-II was used in studies one and two. The DSM-IV version was used in study three. Initially participants were required to complete the self-report personality questionnaire (SCID-PQ) of the SCID-II at baseline. It was followed up by an interview six weeks later which was guided by items affirmed on the SCID-PQ. Clinical judgment ascertained whether the criterion for a PD was satisfied. For this criterion to be satisfied, characteristics must be pathological, persistent and pervasive. The method used to diagnosis individuals was in accordance with instructions for using the SCID-II and enabled personality disorder symptomatology to be based upon clinical contact and a structured clinical interview. Items not affirmed on the SCID-PQ were assumed to be true negatives. However, the interviewer followed up items not affirmed if they had any reason to believe the responses were false negatives.

In studies one and two, the treating clinician assessed personality disorder symptomatology. However, due to personality being an important predictor of outcome in study three, an independent clinician assessed personality disorder symptomatology. To assess inter-rater reliability, within study two, an independent clinician who previously had

no contact with the participants re-interviewed 10% of the participants with the SCID-II. Inter-rater reliability, within study two, assessed by kappa (*k*) for the presence of any personality disorder was .78. Inter-rater agreement for the SCID-II from previous research has been found to be satisfactory (Maffei, et al., 1997) except for histrionic personality traits (Dreessen & Arntz, 1998). Internal consistency coefficients (*k*) from previous research are satisfactory, ranging from .71 to .94 (Maffei, et al., 1997).

2.4.5 Risk Factors

2.4.5.1 Tri-dimensional Personality Questionnaire (TPQ); Temperament and Character Inventory (TCI) (Cloninger, 1987; Cloninger, et al., 1993)

The TPQ is a 100-item true/false self-report questionnaire used to describe temperament, from which three major scales are derived. These are novelty-seeking, harm avoidance, and reward dependence. Cloninger, Pryzbeck, Svrakic, and Wetzel (1994) define temperament as the unconscious, automatic emotional response biases that individuals inherit. This measure was designed to be comprehensive measure of personality. However, Cloninger, et al. (1994) later added another temperament dimension, persistence, and three character dimensions into their theory of personality. Character was defined as individual differences in self-concept, which develop through maturation and learning. The character dimensions are self-directedness, cooperativeness, and self-transcendence. This resulted in the development of the TCI.

The TCI is a true/false self-report questionnaire from which seven major subscales are derived describing temperament and character. It was developed to identify individual differences in normal and abnormal behaviour patterns. The version of the TCI used in study two was a 238-item scale and in study three a 293-item scale (other versions vary in the number of items). The seven factor structure has been replicated in both the general population and in psychiatric patient populations (Cloninger, et al., 1994, Sato, et al., 2001). Internal consistency is satisfactory (Cronbach alpha coefficients range from .68-.82), however, it is weak in the persistence scale (.49; Pelissolo & Lepine, 2000). The TCI, in general, is robust against the state effect of depression in those with mild to moderate depression (Sato, et al., 2001). Hence it is able to characterise the underlying personality structure of individuals with mild to moderate depression.

Both the TPQ and TCI (238- and 293-item) questionnaires were analysed in this thesis, the measures have been scaled so that each measure can be compared across different versions of the TPQ and TCI. The equation used for this is: Scaling number – (score/x) 100 where x is the number or questions in each subscale. The TPQ does not include the character scales. Therefore, only character scales from study two and three will be included in the results. The TPQ and TCI scale means are shown in Table 2.

Table 2

The TPQ and TCI Scale Means

Items	Scale	Study	NS	HA	RD	RD2/PS	SD	C	ST
100	TPQ	One	47.75	67.21	57.63	56.02	-	-	-
238	TCI	Two	51.93	66.92	62.03	53.96	54.53	76.15	33.2
293	TCI	Three	49.12	69.17	65.19	43.32	53.23	77.62	31.86

2.4.5.2 Childhood Sexual Abuse Interview

This is a semi-structured interview created by the Department of Psychological Medicine, it guides the clinician to collect information about the participant's history of childhood sexual abuse. For example, the type of abuse (e.g., touching, intercourse), age when abuse occurred, relationship to perpetrator, personal reactions to the abuse, disclosure and whether they sought any counselling (see Appendix A).

2.4.5.3 Family Psychiatric History Interview

This is a semi-structured interview created by the Department of Psychological Medicine, it guides the clinician to collect information about the participant's relatives (first-degree relatives over 16 years), those they live with (spouse/ partner of at least 6 months) and their family history of mental illness (see Appendix B).

2.4.5.4 Parental Bonding Inventory (PBI) (Parker, et al., 1979)

The PBI is a 25-item self-report questionnaire assessing maternal and paternal behaviour. It is a retrospective judgment of the quality of the participant's relationship with each parent prior to 16 years of age. This scale assesses paternal and maternal parental care (12 items) and protection (13 items). The caring/rejection dimension evaluates the theme of "affection, emotional warmth, empathy, closeness" versus "emotional coldness, indifference and neglect". The protection dimension evaluates "control, overprotection, intrusion, excessive contact, infantilisation" versus "allowance of independence and autonomy"

(Parker, et al., 1979). Higher scores on the care scale represent more positive parental behaviour while higher scores on the overprotection scales represent less positive parental behaviour.

The factors of care and overprotection have been confirmed in clinical and non-clinical populations (see Parker 1990). The PBI has good long-term (10 years) reliability ranging from .56-.72 (Wilhelm & Parker, 1990). Internal consistencies range from .74-.95, with a median alpha coefficient of .89 and a mean alpha coefficient of .88 (Parker, 1989). The median test-re-test reliability reported by Parker (1989) was .80, and the mean was .82. Further data has shown that the PBI assesses the presence of a risk factor for depression, remains stable after depression remits, is consistently associated with depression and predicts the onset of depression (see Ingram & Ritter, 2000). Studies have also failed to show that PBI scores are unduly influenced by the individuals current emotional or depressive state (Brewin, Andrews, & Gotlib, 1993). This is of great importance, it supports the validity of the PBI for individuals experiencing depression.

2.4.6 Summary

A summary of each of the measures used in each of the three studies is presented in Table 3.

Table 3

Measures Utilised in this Thesis From Each Study

	Study One	**Study Two**	**Study Three**
Clinician rated depressive criteria	SCID-P GAF	SCID-P GAF	SCID-P GAF
Clinician rated depressive symptoms	HDRS (17-item) -	HDRS (27-item) MADRS	HDRS (27-item) MADRS
Self-report depressive symptoms and functioning	SCL-90-R - SAS	SCL-90-R DAS SAS	SCL-90-R DAS SAS
Axis I and axis II comorbidity	SCID-P (DSM-III-R) SCID-II (DSM-III-R)	SCID-P (DSM-III-R) SCID-II (DSM-III-R)	SCID-P (DSM-III-R & DSM-V) SCID-II (DSM-IV)
Risk factors	TPQ (100-item) - Family psychiatric history interview PBI	TCI (238-item) Childhood sexual abuse interview Family psychiatric history interview PBI	TCI (293-item) Childhood sexual abuse interview Family psychiatric history interview PBI

2.5 STATISTICAL ANALYSES

All data for the three studies were transferred from a relational database, Paradox (Borland International, 1988) to SPSS (SPSS Inc., 1997) for statistical analyses. Full checking of data was conducted through visually verifying descriptive statistics, by scanning the data set, and continually checking data throughout the analyses. A biostatistician was consulted regularly on all analyses used in this thesis.

Analyses were exploratory. Demographic data was analysed with analyses of variance and general linear modelling (GLM)[1]. The effects of *age*, *gender*, and *gender-by-age interactions* on symptomatology, comorbidity and the occurrence of identified risk factors were analysed. Gender was included in the analyses because, as described above, it is influential in the epidemiology of depression and depression may present differently in men and women. These variables were analysed as follows. Continuous dependent variables were analysed using general linear modelling and binary categorical dependent variables were analysed using step-wise logistic regression. Significant findings from these analyses were further explored as follows. Pearson's correlations (r) were performed to determine the form of significant relationships between age and continuous dependent variables, and analyses of variance were used when age was significantly associated with categorical variables. When a gender-by-age interaction was found, data was split by gender and separate correlations or analyses of variance were used for both men and women.

[1] The GLM procedure provides regression-type analysis for continuous dependent variables incorporating continuous and categorical independent variables. It also allows the investigation of interactions between variables as well as the main effects of individual factors.

CHAPTER 3

RESULTS

Analyses of the demographic variables, clinician rated depressive criteria, clinician rated depressive symptoms, self-reported symptoms and functioning, axis I and axis II comorbidity, and the occurrence of identified risk factors were conducted. Continuous dependent variables were analysed using general linear modelling and Pearson's *r* correlations. Within the text, these results are summarised as *F*-values, degrees of freedom, probability, R^2 and *r* to show the significance values, variance accounted for in age by the dependent variable and the direction of the relationship between age and the dependent variable. Categorical dependent variables were analysed using step-wise logistic regression and analyses of variance. These results are summarised by including the predicted percentage from the regression models, and the means of the absence and presence of dependent variables are presented in tables to show the direction of the relationship between age and the dependent variables.

3.1 Demographic Variables

The demographic variables, such as gender, marital status and ethnicity, across the three studies were analysed to determine if the studies were similar enough to be combined into a larger data set, and analyses investigated whether age was associated with any of the demographic variables. The mean ages within each gender, marital and ethnic group, across each study are presented in Table 4.

Table 4

Demographic Data: Mean Ages Within Each Gender, Marital and Ethnic Group Across the Three Studies

		Study One *N*=110		Study Two *N*=188		Study Three *N*=149	
		M (age)	*SD*	*M* (age)	*SD*	*M* (age)	*SD*
Gender	Women	31.03	9.89	30.08	9.90	34.24	9.35
	Men	31.26	9.37	31.42	10.16	33.88	9.82
Marital status	Single	25.30	6.97	23.84	5.79	28.21	8.32
	Married/living with partner	35.45	8.21	36.89	8.50	37.71	7.76
	Separated/divorced/widowed	38.40	8.52	39.59	7.80	40.08	7.92
Ethnicity	Pakeha	31.34	9.52	30.72	10.07	34.74	9.41
	Maori	25.33	7.77	32.60	9.29	30.25	10.05
	Other	30.40	13.16	23.00	2.00	26.89	6.92

Univariate analyses confirmed that age was significantly associated with marital status (F=148.03, df=2,444, $p\leq$0.01) as would be expected, and study (F=5.86, df=2,444, $p\leq$0.01), but not with gender and ethnicity. Further analyses showed that age, gender, marital status and study all interact. However, the findings appear to be significant due to study three. Study three differs from studies one and two, because it is a psychotherapy study. Participants in study three were predominantly women and were, on average, older. The data from all three studies will be pooled for further analyses because the differences found add to the variability of the sample, just as one would find in any clinical population seeking pharmacotherapy or psychotherapy.

3.2 Clinician Rated Depressive Criteria

3.2.1 Structured Clinical Interview for the DSM-III-R (SCID-P) and the Global Assessment of Functioning Scale (GAF)

Table 5 shows the association between depressive symptomatology on the SCID and age, and gender by age. From this table it can be seen that the occurrence of the majority of the symptoms changed with age. For some symptoms age or age-by-gender had a significant effect but the logistic regression models could not predict symptoms beyond a level of chance. That is, while the variables were significantly related, the equations were unable to strongly predict the presence and absence of the dependent variable from the independent variable(s). This may have been due to small sub-sample sizes. For example, the occurrence of depressed mood and self-mutilation declined with age, whereas, the occurrence of suicidal

ideation or planning increased with age when compared to suicidal attempt, and insomnia increased with age when compared to the presence of hypersomnia. In addition, an age-by-gender interaction was found for the occurrence of fatigue. The occurrence of fatigue declined with age in men and increased with age in women (see Table 6).

The following symptoms were also significantly associated with age and the models were able to predict symptom occurrence. The presence of hypersomnia decreased with age when compared to the absence of sleep difficulties ($p<0.01$; see Table 6). The regression model showed a prediction percentage of 59.4.

As can be seen in Table 6 and Figure 1, the presence of psychomotor retardation declined with age when compared to the presence of psychomotor agitation ($p\leq0.01$). The presence of psychomotor retardation declined with age in men and increased with age in women, when compared to the absence of psychomotor retardation or agitation ($p\leq0.01$). The presence of psychomotor agitation also declined with age in men and increased with age in women when compared to the absence of psychomotor retardation or agitation ($p\leq0.01$). For these three findings, the regression model showed prediction percentages of 58.1, 61.6, and 65.6, respectively.

As can be seen in Table 6, the presence of a suicide attempt declined with age when compared to the absence of suicidal ideation, planning or attempts ($p\leq0.01$). In addition, the presence of suicidal ideation or planning declined with age when compared to the absence of suicidal ideation, planning or attempts ($p\leq0.05$). The regression models for these symptoms showed prediction percentages of 81.4, and 61.6, respectively.

The global assessment of functioning scale (GAF) score was greater in middle-aged participants. The score increased with age ($F=12.12$, $df=1,443$, $p\leq0.01$, 5%, $r=.15$).

Table 5

Comparison of Depression Items in the Structured Clinical Interview for the DSM-III-R (SCID-P) by Age and Age-by-Gender. For Significant Findings, the Direction of the Association with Age is Noted in Parentheses. Where There is an Age-By-Gender Interaction, the Direction for Men is Presented First.

Variable	**Age** ***F***	**Gender x Age** ***F***
Depressed mood (-)	7.19**	-
Fatigue (-) (+)	-	5.95*
Interest	-	-
Weight/ Appetite		
None vs. gain	-	-
None vs. loss	-	-
Gain vs. loss	-	-
Sleep		
Insomnia (both) vs. none	-	-
Hypersomnia (both) vs. none (-)	6.60**	-
Insomnia vs. hypersomnia (not both) (+)	8.27**	-
Psychomotor		
Agitation (both) vs. none (+) (-)	-	7.02**
Retardation (both) vs. none (+) (-)	13.25**	8.52**
Retardation vs. agitation (not both) (-)	7.13**	-
Worthlessness	-	-
Thinking	-	-
Suicide		
None vs. idea/plan (-)	4.87*	-
None vs. attempt (-)	16.19**	-
Idea/plan vs. attempt (+)	9.86**	-
Self-mutilation (-)	10.34**	-
Axis V		
GAF score (+)	12.12**	2.01

Categorical data are blank when neither age or gender by age were significant in the stepwise logistic regression model, and Wald scores are presented for significant findings. * $p \leq 0.05$ ** $p \leq 0.01$.

Table 6

The Mean Ages of Participants Who Presented with a Specific Symptom and Participants Who Did Not. Results are Shown for Men and Women When an Age-by-Gender Interaction was Present.

		Symptom absent *M*	Symptom present *M*
Depressed mood		35.41	31.79
Self-mutilation		33.14	28.92
Insomnia (vs. hypersomnia)		28.23	32.42
Fatigue	Men	34.42	31.79
	Women	31.57	31.91
Hypersomnia (vs. none)		33.07	28.25
Psychomotor retardation (vs. none)	Men	30.57	32.63
	Women	33.49	28.42
Psychomotor retardation (vs. agitation)		33.21	30.40
Psychomotor agitation (vs. none)	Men	30.57	33.06
	Women	33.49	32.76
Suicidal ideation or planning (vs. attempt)		26.00	31.60
Suicidal attempt (vs. none)		33.81	26.00
Suicidal ideation or planning (vs. none)		33.81	31.60

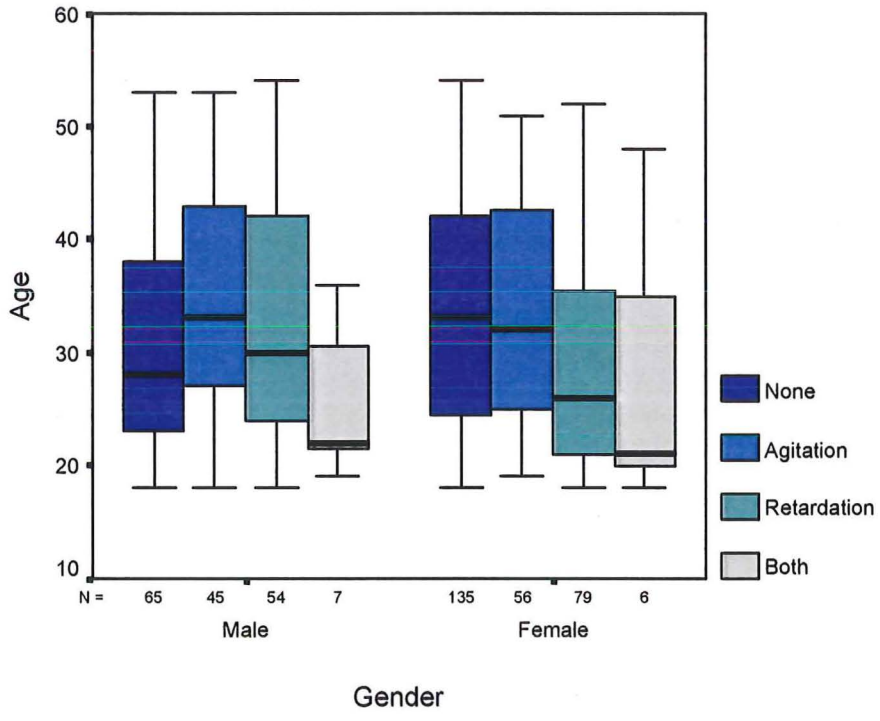

Figure 1. The Interaction Between Age, Gender and Psychomotor Disturbances

3.3 Clinician Rated Depressive Symptoms

3.3.1 Hamilton Depression Rating Scale (HDRS-27/17)

Table 7 shows the association between depressive symptomatology on the HDRS and age, and age by gender. As can be seen, neither the 17- or 27-total scores were significantly related with age, or over half of the symptoms. As was found in the SCID, the occurrence of depressed mood (F=4.98, df=1,443, $p\leq$0.05, 1%, r=-.10), and suicidal ideation or behaviour (F=9.17, df=1,443, $p\leq$0.01, 3%, r=-.13) declined with age. The occurrence of psychomotor retardation also decreased with age, interestingly no age-by-gender interaction was found (F=4.12, df=1,443, $p\leq$0.05, 1%, r=-.10). The occurrence of somatic anxiety increased with age (F=10.30, df=1,443, $p\leq$0.01, 2%, r=.14). In contrast to the SCID, neither fatigue or psychomotor agitation were significantly related to age.

3.3.1.1 Sleep

Analyses revealed significant findings for early, middle and late insomnia, and hypersomnia. The occurrence of early insomnia decreased with age (F=8.74, df=1,443, $p\leq$0.01, 2%, r=-.15), and the occurrence of middle insomnia (F=8.74, df=1,443, $p\leq$0.01, 2%, r=.13), and late insomnia increased with age (F=6.45, df=1,443, $p\leq$0.05, 1%, r=.12).). The occurrence of hypersomnia declined with age (F=6.66, df=1,333, $p\leq$0.05, 2%, r=-.16). These findings are consistent with those of the SCID, with the exception of finding greater early insomnia in younger adults when sub-categories for insomnia were analysed.

Table 7

Comparison of the 17 or 27 Items in the Hamilton Depression Rating Scale (HDRS-27/17) by Age and Age-by-Gender. For Significant Findings, the Direction of the Association with Age is Noted in Parentheses.

Variable	Age *F*	Gender x Age *F*
17-total	0.07	0.31
27-total	0.75	3.06
Depressed mood (-)	4.98*	0.15
Feelings of guilt	0.18	0.23
Suicide (-)	9.17**	1.70
Early insomnia (-)	8.74**	0.54
Middle insomnia (+)	8.74**	2.61
Late insomnia (+)	6.45*	0.28
Work and activities	2.86	0.02
Retardation (-)	4.12*	0.00
Agitation	0.13	0.66
Anxiety-psychic	1.82	0.00
Anxiety- somatic (+)	10.30**	1.68
Somatic- gastrointestinal	1.02	0.05
Somatic- general	0.27	1.17
Genital symptoms	0.69	0.04
Hypochondriasis	0.40	2.64
Weight loss	0.00	0.08
Insight	-	-
Diurnal variation	-	-
Depersonalization	0.52	0.00
Paranoia	3.19	3.67
Obsessions and compulsions	0.07	0.56
Fatigability	1.77	0.67
Social withdrawal	0.72	0.01
Appetite increase	0.03	1.13
Carbohydrate craving	1.34	0.48
Weight gain	0.26	0.74
Hypersomnia (-)	6.66*	1.00
Global evaluation	0.66	0.07

Categorical data are blank when neither age or gender by age were significant in the stepwise logistic regression model, and Wald scores are presented for significant findings. Continuous data are presented as *F*-ratio's. * $p \leq 0.05$ ** $p \leq 0.01$.

3.3.2 Montgomery Asberg Depression Rating Scale (MADRS)

Table 8 shows the association between items on the MADRS with age, and gender by age. In contrast to results on the HDRS and SCID, only two items were significantly related to age. Results showed that the occurrence of inner tension increased with age (F=7.79, df=1,333, $p \leq 0.01$, 2%, r=.14). In addition, the occurrence of reduced sleep increased with age

(F=7.08, df=1,333, $p\leq$0.01, 2%, r=.14), as was found in the SCID. An interesting finding was that suicidal thoughts were not significantly associated with age.

Table 8

Comparison of Montgomery Asberg Depression Rating Scale (MADRS) Items by Age and Age-by-Gender. For Significant Findings, the Direction of the Association with Age is Noted in Parentheses.

Variable	Age *F*	Gender x Age *F*
Apparent sadness	2.71	0.24
Reported sadness	0.87	0.80
Inner tension (+)	7.79**	0.78
Reduced sleep (+)	7.08**	0.22
Reduced appetite	0.15	0.29
Concentration difficulties	0.00	0.99
Lassitude	0.07	0.07
Inability to feel	0.02	0.07
Pessimistic thoughts	0.43	1.15
Suicidal thoughts	2.41	0.10
MADRS total score	0.00	0.00

Continuous data are presented as F-ratio's. * $p\leq$ 0.05 ** $p\leq$ 0.01.

3.3.3 Hopkins Symptom Checklist- Revised (SCL-90-R)

Table 9 shows the association between items on the SCL-90 with age, and gender by age. As can be seen, the majority of the subscales were significantly associated with age. The occurrence of interpersonal sensitivity (F=17.82, df=1,442, $p\leq$0.01, 4%, r=-.21), anger-hostility (F=20.57, df=1,442, $p\leq$0.01, 5%, r=-.23), phobic anxiety (F=16.55, df=1,442, $p\leq$0.01, 3%, r=-.19), paranoid ideation (F=8.64, df=1,442, $p\leq$0.01, 2%, r=-.15), and psychoticism (F=21.00, df=1,442, $p\leq$0.01, 5%, r=-.21), all declined with age. This is in contrast to the lack of significant findings of 'paranoia' on the HDRS.

Only a minority of the depressive symptoms were significantly associated with age. Surprisingly, analyses revealed a significant relationship between age and the depression subscale. Endorsement of the depression subscale declined with age (F=5.62, df=1,442, $p\leq$0.05, 1%, r=-.12). Loss of libido increased with age (F=4.93, df=1,433, $p\leq$0.05, 3%, r=.11), and as was found in the SCID, HDRS and MADRS, the occurrence of suicidal ideation was greater in younger individuals (F=23.38, df=1,440, $p\leq$0.01, 5%, r=-.22). Anhedonia also declined with age (F=7.38, df=1,438, $p\leq$0.01, 1%, r=-.14) , although 'loss of

interest' was not significant on the SCID. The occurrence of hopelessness declined with age (F=8.41, df=1,441, $p\leq$0.01, 1%, r=-.14), although 'pessimistic thoughts' were not significant on the MADRS. Results showed a significant interaction between the 'crying easily' item, age and gender. As shown in Figure 2, the endorsement of crying easily declined with age in women but not men (F=5.79, df=1,442, $p\leq$0.05, 11%, r=-.14). Interestingly, neither anxiety or low energy were significant, although, somatic anxiety and fatigue were significantly related to age on the HDRS.

Table 9

Comparison of Hopkins Symptom Checklist (SCL-90-R) Subscales and SCL-90-R Depression Subscale Items by Age and Age-by-Gender. For Significant Findings, the Direction of the Association with Age is Noted in Parentheses. Where There is an Age-By-Gender Interaction, the Direction for Men is Presented First.

Variable	**Age** ***F***	**Gender x Age** ***F***
Somatisation	0.00	0.05
Obsessive-compulsive	0.71	0.01
Interpersonal sensitivity (-)	17.82**	0.10
Anxiety	0.37	0.24
Anger-hostility (-)	20.57**	1.84
Phobic anxiety (-)	16.55**	0.46
Paranoid ideation (-)	8.64**	0.59
Psychoticism (-)	21.00**	0.41
Depression (-)	5.62*	0.05
Loss libido (+)	4.93*	0.02
Low energy	3.24	0.12
Suicide ideation (-)	23.38**	1.00
Crying easily (+) (-)	0.24	5.79*
Feeling trapped	0.94	0.01
Self-blame	3.79	3.38
Feel lonely	3.86	0.00
Feel blue	0.57	1.53
Worrying	0.45	0.03
Anhedonia (-)	7.38**	0.05
Hopeless (-)	8.41**	0.02
Effort	0.60	1.51
Worthlessness	2.63	0.00

Continuous data are presented as F-ratio's. * $p\leq$ 0.05 ** $p\leq$ 0.01.

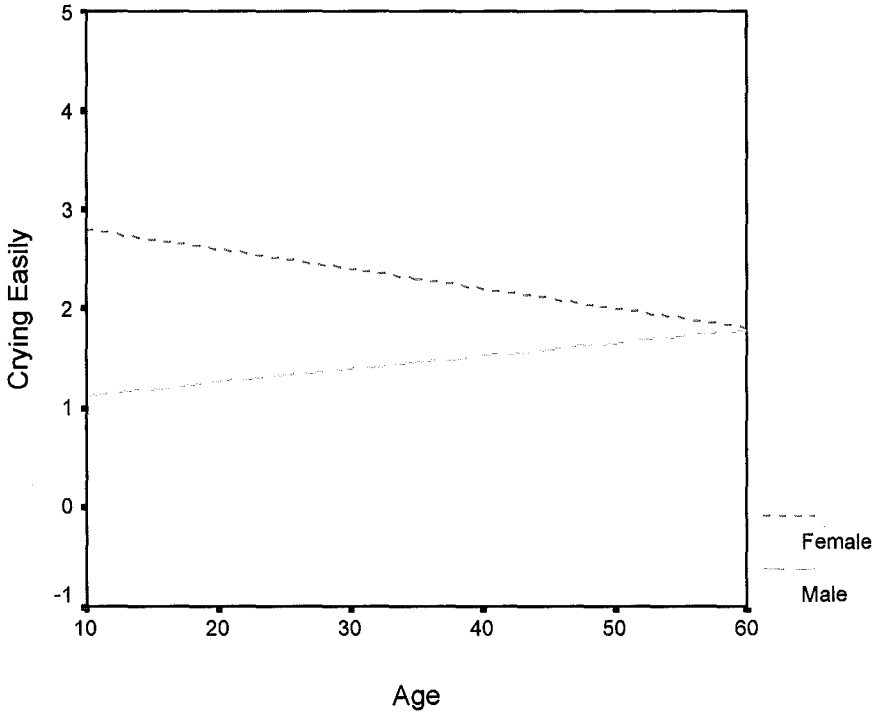

Figure 2. The Interaction Between Age, Gender and 'Crying Easily' on the SCL-90-R

3.3.4 Dysfunctional Attitudes Scale (DAS)

Table 10 shows the association between the DAS total score and age, and gender by age. The DAS total score declined with age (F=8.14, df=1,324, $p\leq$0.01, 2%, r=-.15). Therefore, the total score was greater in younger individuals.

Table 10

Comparison of the Dysfunctional Attitudes Scale (DAS) by Age and Age-by-Gender. The Direction of the Association with Age is Noted in Parentheses.

Variable	Age *F*	Gender x Age *F*
Total Score (-)	8.13**	0.28

Continuous data are presented as F-ratio's. * $p\leq$ 0.05 ** $p\leq$ 0.01.

3.3.5 Social Adjustment Scale (SAS)

Table 11 shows the relationship between items on the SAS and age, and gender by age. Results showed significant relationships between age and three of the four subscales. Scores on the feelings and satisfaction subscale (F=8.98, df=1,442, $p\leq$0.01, 1%, r=-.14), friction subscale (F=20.82, df=1,442, $p\leq$0.01, 6%, r=-.23), interpersonal behaviour subscale

(F= 15.04, df=1,442, $p\leq 0.01$, 3%, r=-.17), and the SAS total score (F=12.62, df=1,442, $p\leq 0.01$, 2%, r=-.17) all declined with age. This is consistent with the decline in interpersonal sensitivity with age on the SCL-90-R and the decline of the GAF score with age on the SCID, however, social withdrawal was not significantly related to age on the HDRS.

Table 11

Comparison of Social Adjustment Scale (SAS) Subscales by Age and Age-by-Gender. For Significant Findings, the Direction of the Association with Age is Noted in Parentheses.

Variable	Age *F*	Gender x Age *F*
Performance	2.75	0.14
Feelings and satisfaction (-)	8.98**	0.13
Friction (-)	20.82**	2.22
Interpersonal behaviour (-)	15.04**	1.64
SAS total score (-)	12.62**	0.14

Continuous data are presented as F-ratio's. * $p\leq 0.05$ ** $p\leq 0.01$.

3.4 Axis I and Axis II Comorbidity

3.4.1 Structured Clinical Interview for DSM-III-R (SCID-P), and the Structured Clinical Interview for DSM-III-R, Axis II Personality Disorders (SCID-II).

Table 12 shows the association between axis I and axis II psychopathology with age, and age-by-gender. As can be seen, only one axis I comorbid disorder and approximately half of the axis II comorbid personality disorders were significantly related to age. For some symptoms age or age-by-gender had a significant effect but the logistic regression models could not predict symptoms beyond a level of chance. For example, the occurrence of current alcohol or drug dependence, schizotypal PD, borderline PD, and antisocial PD declined with age. In addition, the occurrence of both an obsessive-compulsive PD and paranoid PD diagnosis declined with age in men and increased with age in women (see Table 13).

However, general linear modelling indicated that both the total number of personality disorder *diagnoses* (F=4.92, df=1,417, $p\leq 0.05$, 6%, r=-.11) and the total number of personality disorder *symptoms* (F=10.94, df=1,417, $p\leq 0.01$, 7%, r=-.16), decreased with age.

Table 12

Comparison of the Structured Clinical Interview for DSM-III-R (SCID-P) and the Structured Clinical Interview for DSM-III-R, Axis II Personality Disorders (SCID-II), by Age and Age-by-Gender. For Significant Findings, the Direction of the Association with Age is Noted in Parentheses. Where There is an Age-By-Gender Interaction, the Direction for Men is Presented First.

Variable	**Age** ***F***	**Gender x Age** ***F***
Axis I		
MDD		
Lifetime	-	-
Current	-	-
Bipolar II		
Lifetime	-	-
Current	-	-
Alcohol or Drug Dependence		
Lifetime	-	-
Current (-)	4.41*	-
Anxiety Disorder		
Lifetime	-	-
Current	-	-
Eating Disorder		
Lifetime	-	-
Current	-	-
Axis II		
Avoidant PD	-	-
Dependent PD	-	-
Ob-Compulsive PD (+) (-)	-	14.72**
Paranoid PD (+) (-)	-	5.48*
Schizotypal PD (-)	5.06**	-
Schizoid PD	-	-
Histrionic PD	-	-
Narcissistic PD	-	-
Borderline PD (-)	10.54**	-
Antisocial PD (-)	4.77*	-
Other	-	-
Total # of Diagnoses (-)	4.93*	0.03
Total # of Symptoms (-)	10.95**	0.28

Categorical data are blank when neither age or gender by age were significant in the stepwise logistic regression model, and Wald scores are presented for significant findings. Continuous data are presented as *F*-ratio's. * $p \leq 0.05$ ** $p \leq 0.01$.

Table 13

The Mean Ages of Participants Who Were Diagnosed with a Comorbid Psychiatric Disorder and Participants Who Were Not. Results are Shown for Men and Women When an Age-by-Gender Interaction was Present.

		Disorder absent *M*	Disorder present *M*
Current alcohol or drug dependence		32.09	28.93
Schizotypal PD		32.31	26.27
Borderline PD		32.79	28.42
Antisocial PD		32.34	27.38
Obsessive-compulsive PD	Men	31.47	36.21
	Women	32.27	28.13
Paranoid PD	Men	32.00	33.03
	Women	32.43	28.83

3.5 Risk Factors

3.5.1 Temperament and Character Inventory (TCI) and the Tri-dimensional Personality Questionnaire (TPQ)

Table 14 shows the association between the subscales on the TCI or TPQ and age, and gender by age. The majority of the subscales were associated with age. The novelty-seeking (F=46.90, df=1,432, $p\leq$0.01, 11%, r=-.34), and harm avoidance (F=4.08, df=1,432, $p\leq$0.05, 2%, r=-.09) scale scores declined with age, whereas the self-directedness (F=9.26, df=1,322, $p\leq$0.01, 4%, r=.18), and cooperativeness (F=11.03, df=1,322, $p\leq$0.01, 8%, r=.18) scale scores increased with age. A significant interaction between the persistence subscale, age and gender was evident (F=5.39, df=1,432, $p\leq$0.05, 3%, r=.18). Specifically, persistence scale scores increased with age in women but not men (see Figure 3).

Table 14

Comparison of the Temperament and Character Inventory (TCI) Scales and the Tri-dimensional Personality Questionnaire (TPQ) Scales by Age and Age-by-Gender. For Significant Findings, the Direction of the Association with Age is Noted in Parentheses. Where There is an Age-By-Gender Interaction, the Direction for Men is Presented First.

Variable	Age *F*	Gender x Age *F*
Novelty-seeking (-)	46.90**	2.88
Harm Avoidance (-)	4.08*	0.44
Reward Dependence	0.48	1.61
Persistence (-) (+)	2.21	5.39*
Self-directedness (+)	9.26**	0.39
Cooperativeness (+)	11.03**	0.01
Self-Transcendence	0.00	0.02

Continuous data are presented as *F*-ratio's. * $p \leq 0.05$ ** $p \leq 0.01$.

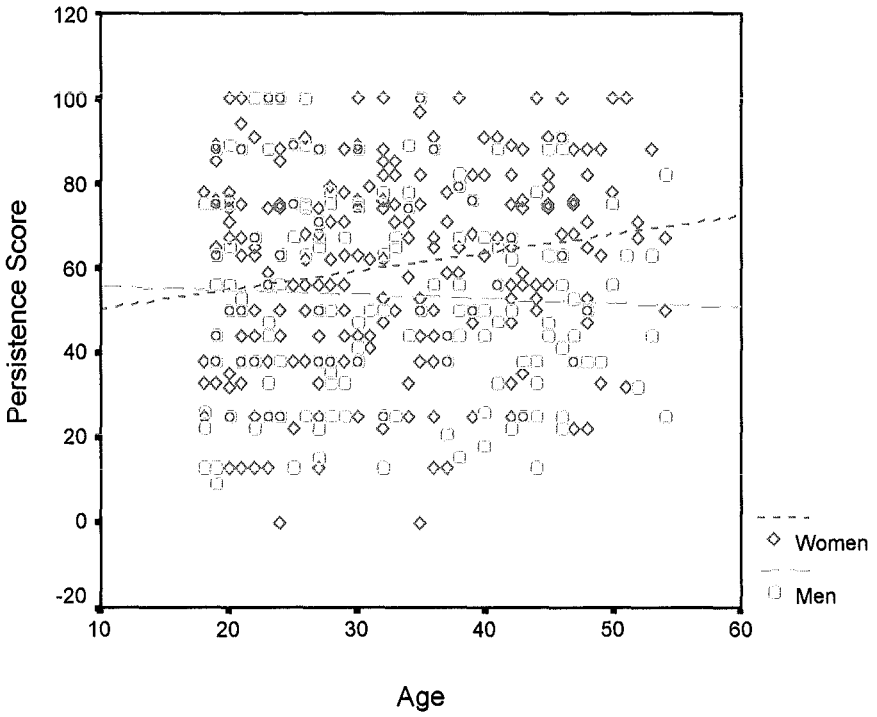

Figure 3. The Interaction Between Age, Gender and the Persistence Scale on the TCI/TPQ

declined with age in men and increased with age in women (see Table 16). However, the logistic regression model could not predict this beyond a level of chance.

The logistic regression model *was* able to predict the interaction between familial affective disorders, age and gender (64.8%). As can be seen in Table 16 and Figure 4, the rate of familial affective disorders declined with age in men and increased with age in women.

Table 15

Comparison of Family History Interview Items by Age and Age-by-Gender. The Direction of the Association with Age is Noted in Parentheses, the Direction For Men Is Presented First.

Variable	**Age** *F*	**Gender x Age** *F*
Alcohol Dependence (-) (+)	5.62*	7.69**
Affective Disorders (-) (+)	15.83**	8.19**

Categorical data are blank when neither age or gender by age made it into the stepwise logistic regression model, and Wald scores are presented for significant findings. * $p \leq 0.05$ ** $p \leq 0.01$.

Table 16

The Mean Ages of Participants Whose FDR's Were Diagnosed With Alcohol Dependence or Affective Disorders and Those Who Were Not.

		Disorder absent *M*	Disorder present *M*
Familial alcohol dependence	Men	32.53	31.29
	Women	30.91	34.43
Familial affective disorders	Men	32.39	32.13
	Women	28.45	33.83

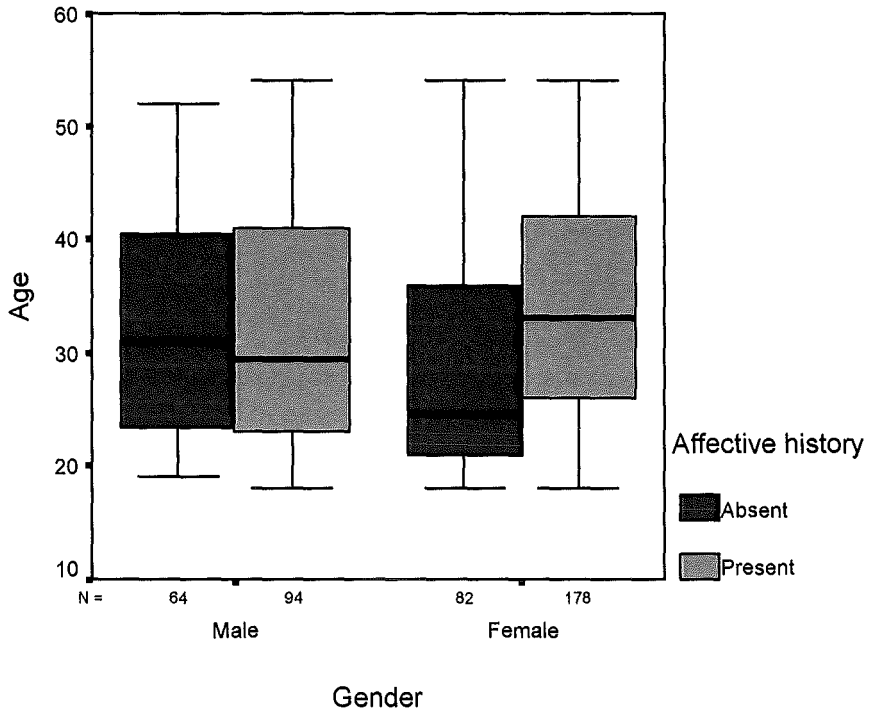

Figure 4. The Interaction Between Age, Gender and Familial Affective History

3.5.3 Childhood Sexual Abuse Interview

No significant findings were found between childhood sexual abuse and age, and gender by age.

3.5.4 Parental Bonding Inventory (PBI)

Table 17 shows the association between the four scales on the PBI and age, and gender-by-age. The majority of the subscales were significantly related to age or showed an age-by-gender interaction. The degree of reported maternal care declined with age (F=10.36, df=1,427, $p\leq$0.01, 3%, r=-.17). As can be seen in Figure 5, the degree of reported maternal protection increased with age in women but not men (F=4.63, df=1,427, $p\leq$0.05, 2%, r=.16). As can be seen in Figure 6, the degree of reported paternal care increased with age in men and decreased with age in women (F=10.66, df=1,408, $p\leq$0.01, 3%, r=-.17).

Table 17

Comparison of the Parental Bonding Inventory Subscales by Age and Age-by-Gender. For Significant Findings, the Direction of the Association with Age is Noted in Parentheses. Where There is an Age-By-Gender Interaction, the Direction for Men is Presented First.

Variable	**Age** ***F***	**Gender x Age** ***F***
Maternal Care (-)	10.36**	1.75
Maternal Protection (-) (+)	1.56	4.63*
Paternal Care (+) (-)	0.06	10.66**
Paternal Protection	2.37	1.50

Continuous data are presented as *F*-ratio's. * $p \leq 0.05$ ** $p \leq 0.01$.

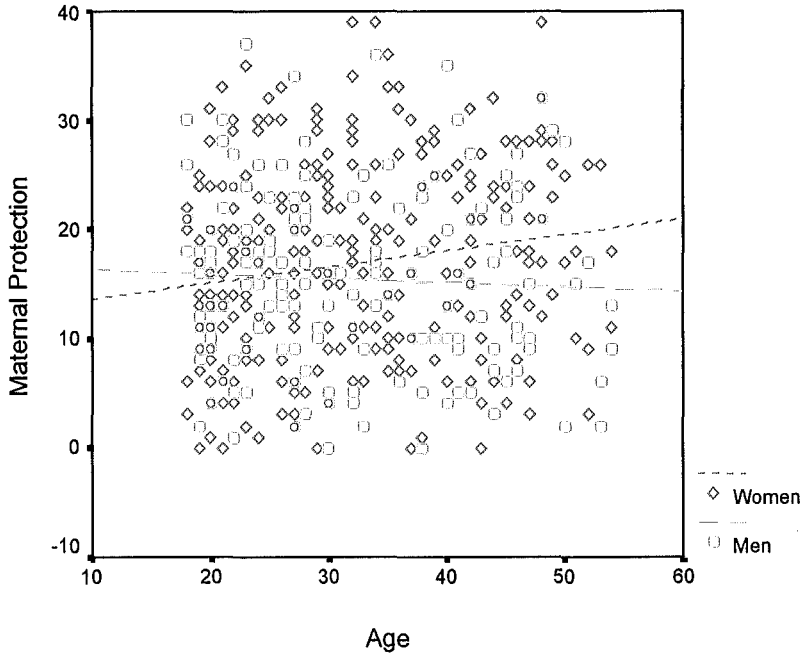

Figure 5. The Interaction Between Age, Gender and the Maternal Protection Subscale on the PBI

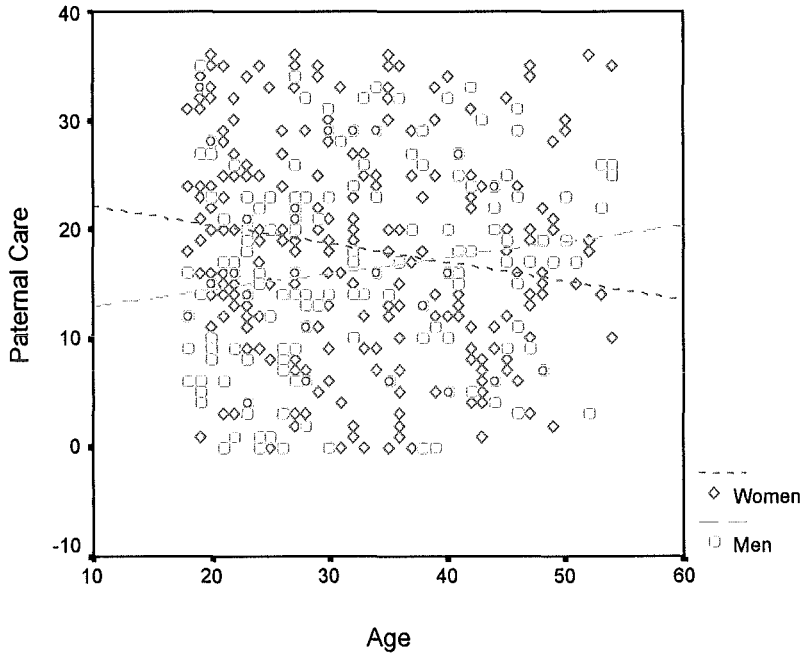

Figure 6. The Interaction Between Age, Gender and the Paternal Care Subscale on the PBI

CHAPTER 4

DISCUSSION

The aim of this thesis was to investigate age-related changes in symptomatology, comorbidity and the occurrence of identified risk factors for depression during early and middle adulthood. A number of these were significantly related to age and are discussed in detail below. First, however, it is noteworthy that the amount of variance accounted for in age by a number of the dependent variables individually was often low. This limits the clinical utility of these results because they are not highly robust, however, as will be discussed, the results are important because they highlight a number of areas that require further attention.

4.1 Clinician Rated and Self-reported Depressive Symptoms

4.1.1 Depressed Mood and Anhedonia

The occurrence of depressed mood declined with age on the SCID, and although it was not predicted beyond a level of chance, depressed mood also significantly declined with age on the HDRS. This is in contrast to number of previous studies (Carlson & Kashini, 1988; Cooper & Goodyer, 1993; Wallace & Pfohl, 1995) where depressed mood occurred consistently across childhood, adolescence and adulthood. A possible reason why depressed mood on the SCID could not be predicted adequately by the regression model, although it was significant on the HDRS, may be due to criterion measures being less sensitive than continuum measures.

The occurrence of anhedonia declined with age on the SCL-90-R, although 'loss of interest' was not significantly related to age on the SCID. The former finding is inconsistent with previous literature stating that anhedonia is stable across adulthood (Garvey & Schaffer, 1994; Wallace & Pfohl, 1995), while the latter is supportive. In addition to being partially inconsistent with adult literature, neither the changes demonstrated in depressed mood or anhedonia on the SCID reflect adolescent literature. There is no readily apparent reason for the greater occurrence of depressed mood and anhedonia in younger individuals.

4.1.2 Anger

The occurrence of anger declined with age on the SCL-90-R suggesting that younger adults may experience anger more often while depressed, than middle-aged adults. Due to the

lack of literature surrounding anger, research on irritability will be discussed. This decline in anger is somewhat consistent with research by Wallace and Pfohl (1995). They found that irritability declined with age in adult women. Although, others disagree and have found that irritability occurs at similar rates across adulthood (Garvey & Schaffer, 1994). By extending the discussion to adolescence, the greater occurrence of anger in young people may be consistent with the fact that irritability is noted within the DSM-IV-TR (APA, 2000) to be more common in adolescents than depressed mood. Though to complicate the issue further, young adults in this sample also demonstrated greater occurrences of depressed mood than middle-aged adults.

4.1.3 Fatigue

The occurrence of fatigue declined with age in men but increased with age in women on the SCID, although, it was not predicted beyond a level of chance. Conversely, age was not associated with fatigue on the HDRS, 'loss of energy' on the SCL-90-R, or lassitude on the MADRS. Overall these findings indicate that fatigue/ loss of energy/ lassitude occur at a similar rate during young and middle adulthood despite a weak association on the SCID. This supports Garvey and Schaffer's (1994) results that loss of energy is not influenced by age. It is somewhat problematic comparing loss of energy, fatigue and lassitude, however, these items are very similar.

4.1.4 Anxiety Symptoms

Surprisingly, the presence of *somatic* anxiety increased with age on the HDRS. This is in opposition to literature claiming that somatic anxiety occurs at a stable rate across adulthood (Garvey & Schaffer, 1994; Wallace & Pfohl, 1995). However, others have found that somatic anxiety is more common in children than adolescents (Cooper & Goodyer, 1993). Hence based on previous research and the current findings, it appears that somatic anxiety is not common in younger adults or adolescents. The occurrence of *phobic* anxiety decreased with age on the SCL-90-R. This may provide some support for the finding that individuals with childhood-onset MDD present with greater social and simple phobias (Alpert, et al., 1999). *Psychic* anxiety on the HDRS was not significantly related to age. This finding is consistent to Garvey and Schaffer's (1994) research, but not other research which has found that psychic anxiety increased with age in adult women (Wallace & Pfohl, 1995). Overall, somatic anxiety increased, phobic anxiety decreased and psychic anxiety was stable across young and middle adulthood. This may also provide evidence for Kovacs (1996) claim

that rates of anxiety disorders do not vary significantly across age. These findings suggest that the rates of anxious symptoms may be stable also. However, as discussed earlier, it appears that anxiety is expressed differently at different ages.

4.1.5 Psychosis

Previous literature has suggested that the occurrence of delusions increased with age (Carlson & Kashini, 1988). However, the occurrence of paranoid ideation and psychoticism declined with age on the SCL-90-R in the present study. One explanation for this difference is that perhaps the occurrence of delusions increases with age but the occurrence of hallucinations decreases with age. Akin to previous research, the findings of this thesis were not consistent. Paranoia was not associated with age on the HDRS in this thesis. This provides some support for previous studies that have also shown no association of psychosis to age (Garvey & Schaffer, 1994; Wallace & Pfohl, 1995). An explanation of the mixed findings may be found within the measure properties. The SCL-90-R is a self-report measure and the HDRS is a clinician rated scale, this is discussed in detail below.

4.1.6 Suicidal Behaviour, Ideation and Self-harm

As mentioned, a well documented finding is the significant increase of suicidal behaviour in adolescence which then declines across adulthood (Cooper & Goodyer, 1993; Wallace & Pfohl, 1995; Fergusson, et al., 2000). This thesis found that the presence of a suicide attempt declined with age on the SCID, the occurrence of suicidal planning or behaviour declined with age on the SCID and HDRS, and the presence of suicidal ideation declined with age on the SCID, HDRS and SCL-90-R. However, suicidal thoughts were not significantly related to age on the MADRS. Overall this thesis provides fairly solid results suggesting that the occurrence of suicidal attempts, behaviour and ideation declines with age. This supports findings from Wallace and Pfohl (1995) who demonstrated that suicidal ideation declined across adulthood. Yet, this is in contrast to some studies claiming that the occurrence of attempts (Garvey & Schaffer, 1994) and ideation (Carlson & Kashini, 1988; Garvey & Schaffer, 1994) is consistent across adulthood. By comparing these findings to research with adolescents, these findings do not appear so surprising. Based on previous research and the current findings, young adults and adolescents both exhibit greater suicidal behaviour than middle-aged adults.

These findings provide some support for Malone, et al.'s (1995) conclusions that the first three months after the onset of an MDE and the first five years after the lifetime onset of MDD represent the highest-risk period for attempted suicide. Based on this theory and the fact that adolescence and young adulthood are peak times of onset of MDE's, the results of this thesis are expected.

An interesting findings was that gender was not significantly associated with suicidal behaviour and ideation. This is in opposition to previous research reporting greater rates of suicidal ideation and attempts in women than men (Fergusson, et al., 2000).

The presence of self-mutilation declined with age on the SCID, although, it was not predicted beyond a level of chance. Though it appears consistent with the above findings, there were no other measures of self-mutilation hence, there is weak evidence.

4.1.7 Psychomotor Activity

The presence of psychomotor retardation declined with age in women but increased with age in men on the SCID. Although in the HDRS, there was no association with gender, the overall occurrence declined with age. These findings contrast previous research showing that psychomotor retardation was stable across adulthood (Garvey & Schaffer, 1994; Wallace & Pfohl, 1995). Aside from the gender difference, the current findings suggest that younger adults experience more psychomotor retardation than middle-aged adults. It appears that young adults may again be similar to adolescents because psychomotor retardation is more common in adolescents than children (Carlson & Kashini, 1988).

Gender was also influential in the presence of psychomotor agitation. The presence of psychomotor agitation also declined with age in women but increased with age in men on the SCID. This is in opposition to previous findings of agitation increasing with age in women but not men (Wallace & Pfohl, 1995). However, no association with age was found on the HDRS in the current study. Which again is in opposition to previous findings that agitation was more common in those 40-80 years of age (Garvey & Schaffer, 1994). These findings are mixed and are contrary to previous research.

4.1.8 Vegetative Symptoms

The occurrence of insomnia increased with age on the SCID, although, it was not predicted beyond a level of chance. Yet, the occurrence of reduced sleep on the MADRS also increased with age. This is inconsistent with previous research claiming that the rate of insomnia does not vary across adulthood except for late insomnia in those 40 to 80 years of age (Garvey & Schaffer, 1994; Wallace & Pfohl, 1995). However, it may be consistent with literature from adolescents where sleep difficulties occur less frequently than in adults (Dahl, 1996; Kaufman, et al., 2001). Hence, younger adults may have a similar expression of depression as adolescents. This appears to provide support for Benca, et al.'s (1992) theory that age may enhance the effects of depression on sleep where the occurrence of insomnia increases with age.

More specific findings were revealed when sub-categories of insomnia were analysed. Consistent with hypotheses, the occurrence of early insomnia declined with age and the occurrence of late insomnia increased with age on the HDRS. Surprisingly, the occurrence of middle insomnia also increased with age. These findings support Garvey and Schaffer's (1994) comment that in older individuals, the natural propensity to wake early is amplified by the depression. The increase in middle insomnia may also be explained by normal developmental changes. As adults age, they experience less slow-wave sleep and a diminished sleep efficacy (Dahl, et al., 1992). This may contribute to an increase in middle insomnia. However, this theory does not provide an explanation of why early insomnia is more common in younger adults. Previous research from depressed adolescents shows consistent evidence of prolonged sleep latency with few other sleep changes occurring during depression (Benca, et al., 1992; Dahl, 1996; Dahl, et al., 1996). Hence young adults in this thesis appear to be more similar to adolescents, than middle-aged adults, as described in the literature.

The occurrence of hypersomnia decreased with age on the SCID and HDRS. This is inconsistent with previous findings that hypersomnia increased across the lifespan until older adulthood (Ryan et al., 1987; Garvey & Schaffer, 1994; Kovacs, 1996). Again by looking to normal developmental differences, a possible explanation is provided. Normal adolescents experience greater sleepiness during the day (Dahl, et al., 1992). Young adults in this sample may be presenting with enhanced normal developmental sleep changes as seen in adolescents.

4.1.9 Dysfunctional Cognitions

The total DAS score declined with age suggesting that there is a greater frequency of dysfunctional attitudes in younger individuals. This is consistent with previous research showing that individuals who were younger at admission and age of depression onset presented with elevated levels of dysfunctional cognitions (Norman, et al., 1988). Although, as suggested by Luty, et al. (1999) this may be due to the duration of depression in younger individuals, not age. Yet, older individuals in the present study showed lower DAS scores and one would expect a better prognosis for younger individuals because many of them would be experiencing their first episodes of depression. In addition, the occurrence of hopelessness declined with age on the SCL-90-R, but pessimistic thoughts were not significantly associated with age on the MADRS.

4.1.10 Loss of Libido and Crying

Reports of loss of libido increased with age on the SCL-90-R. This is opposite to findings by Garvey and Schaffer (1994), though, they were even surprised that loss of libido was more common in younger individuals. The endorsement of the 'crying easily' item on the SCL-90-R declined with age in women but was not significant for men. Previous research shows mixed findings on this symptom. Garvey and Schaffer (1994) found that crying was not associated with age for either men or women. Yet, Wallace and Pfohl (1995) found that crying declined with age in men but was not significantly related to age in women. These findings contrast the present findings. Wallace and Pfohl assessed crying with the Beck Depression Inventory (BDI; Beck, Steer, & Brown, 1987) and this thesis relied on the SCL-90-R which may have partially contributed to the different result.

4.2 Clinician Rated and Self-Reported Psychological, Social and Occupational Functioning

Interestingly, scores on the feelings and satisfaction, friction and interpersonal behaviour subscales on the SAS declined with age suggestive of greater social impairment in younger individuals. A limitation of this finding is that young individuals may not be functioning in all of the roles. Therefore, scores may not fully reflect impairment (Weissman, Scholomskas, & John, 1981). The occurrence of interpersonal sensitivity also declined with age on the SCL-90-R. Yet, social withdrawal was not significantly related to age on the HDRS. The GAF score increased with age suggestive of a greater level of psychological, social or occupational impairment in younger individuals. The majority of these results

suggest greater psychological, social and occupational impairment in younger adults than middle-aged adults. This is consistent with Luty, et al., (2002) who also found that younger individuals showed greater SAS total scores, friction scores and interpersonal behaviour scores indicating social impairment associated with depression declines with age in those 18 to 64.

Young adulthood is a time of change and significant developmental changes. Perhaps experiencing a MDE at a younger age disrupts development (e.g., emotional regulation, socialisation, decision-making skills) resulting in greater impairment. It is noteworthy that other symptoms that decline with age may contribute to the greater impairment such as dysfunctional cognitions, suicidal behaviour, anger, depressed mood, anhedonia, and personality disorders.

As mentioned earlier, research indicates that individuals with depression over-report poor social adjustment during the acute illness phase (Morgado, et al., 1991). However, the influence of age was not analysed. Therefore, it is unknown whether these findings of greater social impairment in younger individuals are due to reporting bias or not.

4.3 Summary

The change in symptomatology with age demonstrated in the current study provides some support for the hypothesis that young adult depression may be more similar to adolescent depression than it is to depression during middle adulthood. Findings that appeared to be more similar to the research from adolescents than adults included findings for anger, suicidal ideation, planning and attempts, hypersomnia and early insomnia. The results for a number of the other symptoms were mixed such as anhedonia, anxiety, psychosis, psychomotor retardation, and agitation.

An interesting finding observed from the results is the influence of the type of measure on the findings. It appears that less symptoms declined with age when they were assessed with the clinician rated scales (i.e., HDRS & MADRS), than the self-report measures (i.e., SCL-90-R, DAS, SAS). Previous research has found discrepancies between self-report and clinician ratings (Carroll, Fielding, & Blashki, 1973; Piersma & Boes, 1995). Some authors have shown that the disagreement was greatest for psychomotor disturbance, decreased concentration, and indecisiveness (Zimmerman, Coryell, Wilson, & Corenthal,

1986) whereas, others report greatest disagreement for psychological anxiety, somatic anxiety, general somatic symptoms, and agitation, and greatest agreement came from items of restricted meaning such as insomnia, weight loss, and suicide (Carroll, et al., 1973). Carroll, et al. (1973) concluded that self-ratings were useful for detecting the presence of symptoms of depression, but not for quantifying their severity because patients do not have the clinical perspective of physicians for rating the severity of their distressing symptoms. They also concluded that the discrepancy between self-report measures and clinician rated scales became greater with the greater severity of depression. Finally, the authors discussed the fact that psychomotor retardation, difficulty concentrating, and indecisiveness are common in individuals with depression. Therefore, completing self-report measures can be time-consuming and difficult, therefore clinician rated scales are more appropriate measures when assessing individuals with depression.

The results from this study appear to support some of these previous findings. It appears that either younger individuals report greater symptomatology and impairment or middle-aged individuals report less symptomatology and impairment, on self-report measures than clinician rated scales. In addition, the endorsement of the depression subscale on the SCL-90-R declined with age which is surprising considering all participants were diagnosed with a MDE. Previous studies have found that the discrepancies between self- and clinician's assessments of depressive symptoms were not linked to age (Berard, Boermeester, Hartman, & Rust, 1997; Corruble, Legrand, Zvenigorowski, Duret, & Guelfi, 1999). However, it appears that there were age-related biases on the self-report and clinician rated scales in the current study. It is likely that these reporting biases contributed to the mixed results.

With regard to gender, research has shown that females are assessed more accurately by self-report measures and clinician rated scales than males because males tend to mask their depressive symptoms more than females (Berard, et al., 1997). However, the current study did not find many gender-by-age interactions suggesting that gender did not bias the results of the current study. With regard to the clinical interviews in the current study, positive and negative relationships with age were found suggesting that age did not bias these findings.

4.4 Axis I and Axis II Comorbidity

4.4.1 Axis I Comorbidity

Age showed a significant association with current alcohol or drug dependence but not with lifetime dependence. The occurrence of a diagnosis declined with age, although, it was not predicted beyond a level of chance, which, may have been due to the low number (n=25) of individuals with this diagnosis. This trend is consistent with findings by Alpert, et al. (1999) which indicated that alcohol abuse and/or dependence occurred more in individuals with childhood-onset MDD than adult-onset MDD. The authors also found differences between childhood- and adult-onset MDD in the expression of anxiety disorders. However, the anxiety disorders were combined in this thesis because anxiety disorders are often comorbid and listing the rates for specific anxiety diagnoses overestimates the number of cases (see Kovacs, 1996). While significant findings may have been masked, overestimation was prevented. Age was not associated with the occurrence of other axis I diagnoses including MDD, Bipolar II, anxiety disorders, eating disorders. This appears to be consistent with the claim that overall rates of comorbidity do not differ across the lifespan (Kovacs, 1996).

4.4.2 Axis II Comorbidity

Age showed a significant association with schizotypal PD, borderline PD, antisocial PD, and in addition to age, gender also showed an association with obsessive-compulsive PD, and paranoid PD. The presence of schizotypal, borderline and antisocial PD all declined with age. The occurrence of obsessive-compulsive and paranoid PD's declined with age in women but increased with age in men. Yet again, possibly due to small numbers, these diagnoses could not be predicted beyond a level of chance. These trends appear to be distinctly different from previous research by Fava, et al., (1996) who found a higher prevalence of avoidant, histrionic, narcissistic, and borderline PD's in individuals with early-onset depression.

Although individual diagnoses were not strongly predicted and were not consistent with previous research, age was able to predict part of the variance in the total number of personality disorder *diagnoses* and the total number of personality disorder *symptoms*. Again these both declined with age suggesting that personality disorders and symptoms are more common in young adults than middle-aged adults. Possible explanations have been posited by Fava, et al. to explain the differences between individuals with early- and late-onset depression which may or may not be applicable. Firstly, this relationship between early-onset

depression and personality disorders may be due to a longer protracted course of illness. Secondly, early- and late-onset (and in this case young and middle-aged adult depression) may be distinctly different. Finally, PD's may place individuals at risk for depression through interpersonal life events (Fava, et al., 1996) or dysfunctional attitudes (Ilardi & Craighead, 1999). The current findings appear to be consistent with previous research that demonstrated that individuals with depression with a concurrent PD, also have an earlier age of onset for their first episode (Pfohl, Stangl, & Zimmerman, 1984). However, literature also suggests that individuals with depression and a concurrent PD also experience a longer duration of the depressive episode and recurring depressive episodes (Shea, et al., 1987). Based on these findings, middle-aged adults should not have shown less PD diagnoses or symptoms.

Some suggest that while individuals are depressed, they may rate themselves as significantly more impaired (e.g., dependent, labile, irritable) than when their depression has abated (see Speier, et al., 1995). Using information obtained during the study baseline may be biased, however, this bias should be consistent across ages.

4.5 Risk Factors

4.5.1 Dimensions of Personality

The scores of novelty-seeking and harm avoidance[1] declined with age suggesting that these personality traits characterise younger individuals with depression more than middle-aged adults. The scores from the persistence[2] subscale increased with age in women but were not significantly associated with age in men. The scores from the self-directedness and cooperativeness[3] subscales increased with age suggesting that these personality traits characterise middle-aged adults with depression as opposed to younger adults.

Three of the four temperament factors were associated with age. Svrakic, et al. (1996) describe temperament as moderately heritable, and moderately stable throughout life. Hence, discovering developmental changes in the temperament factors, of novelty-seeking, harm avoidance and persistence, is not completely unexpected. However, Svrakic, et al. also state that it is unlikely that the dynamics of psychobiological systems are understandable in terms

[1] Novelty seeking is the tendency to be excitable, exploratory, enthusiastic, and impulsive, whereas, harm avoidance is the tendency to be cautious, tense, apprehensive, and pessimistic.
[2] Persistence is the tendency to be hard-working, stable, and industrious.
[3] Individuals high in self-directedness are highly responsible, purposeful, resourceful and self-accepting, whereas individuals high in cooperativeness are empathetic towards others, helpful, and compassionate.

of linear deterministic relationships. Hence, to infer a linear association between age and these factors is impractical, yet, there is obviously some association with age.

As found within the general population by Cloninger, et al (1993), self-directedness and cooperativeness increased with age, and self-transcendence was not significantly related to age. Also consistent with Cloninger et al's findings, self-transcendence and self-directedness were not associated with gender. However, Cloninger et al. found higher scores of cooperativeness in women than men, though the present study did not. Therefore, similar developmental changes in character dimensions occurred in the current sample of individuals with depression, as individuals in the general population.

4.5.2 Familial History of Affective Disorders and Alcohol Dependence

The rate of familial affective disorders declined with age in men but increased with age in women. The decline found in men is consistent with the majority of literature in this field that individuals with an earlier age of onset have higher familial loading of affective disorders in FDR (Weissman, et al., 1984; Neuman, et al., 1997; Lyons, et al., 1998; Wickramarantne & Weissman, 1998; Klein, et al., 2001). However, the influence of gender has not been a significant contributor within the literature. Some investigators suggest that depression may be more heritable in boys than girls (Eaves, et al., 1997, Eley & Stevenson, 1999), whereas others argue that the heritability of depression is greater in women than men (Kendler, Gardner, Neale, & Prescott, 2001). Even so, this does not explain the change in the rate of familial affective disorders with age. The current findings are also inconsistent with Kendler, et al. (1999) who found a similar familial lability of MDD in men and women.

The rate of familial alcohol dependence also declined with age in men but increased with age in women. Yet again, possibly due to the small number of participants in the sub-sample, the rates of familial alcohol dependence could not be predicted beyond a level of chance. Rende, et al. (1997) found that the FDR's of early-onset (< 20) probands had increased rates of alcoholism, when compared to the FDR's of adult-onset probands, only if they also had MDD. Yet again, the gender difference is unexplained within previous research.

4.5.3 Childhood Sexual Abuse

No significant association was found between the occurrence of childhood sexual abuse and age. This is somewhat consistent with findings by Jaffee, et al. (2002) where CSA

was a risk factor for juvenile- (11, 13 and 15 years) and young adult-onset (18, 21, and 26 years) MDD. Therefore, CSA appears to be consistently reported during adolescence, early and middle adulthood.

4.5.4 Parental Bonding

The degree of maternal care reported on the PBI declined with age, maternal overprotection increased with age in women but it was not significantly related to age in men, and paternal care declined with age in women and increased with age in men. Age was not significantly associated with paternal protection. Age had not been a significant influence on PBI scores in previous research. Claims have been made that the reporting of parental attitudes does not change over time as individuals become further removed from childhood (Parker, et al., 1979) and that reports of childhood histories are stable over time and do not fluctuate with mood (Gotlib, Mount, Cordy, & Whiffen, 1988). The interactions between age and gender discovered on the PBI, in the current study, have not been reported before.

The degree of maternal care appeared to be greater in younger adults than middle-aged adults, both men and women. Perhaps, low maternal care places individuals at risk for developing depression later in life, or perhaps abnormal early interactions with ones mother results in maladaptive patterns of coping (e.g., coping with negative affect) that may predispose individuals to depression (see Birmaher, et al., 1996) and possibly recurring depression.

Maternal overprotection was only related to age in women. This is somewhat consistent with research which shows that women with depression tend to report low caring and high over protection in their mothers (Parker, 1983). Yet, middle-aged women appeared to report greater overprotection than younger women. This is not explained in the literature. Again, perhaps maternal overprotection is associated with late-onset depression in women, recurring depression, or the severity of depression which could all contribute to greater reports in middle-aged women.

The degree of reported paternal care was greater in younger women than middle-aged women and greater in middle-aged men than younger men. Perhaps low paternal care places man at risk of early-onset depression and women at risk of late-onset depression. This

interaction may be linked to paternal depression and the familial loading of affective disorders in younger men found in the current study.

In contrast to research by Parker, et al., (1979), it appears that the reports of these dimensions may change with age. Reports are retrospective and individuals may take a different perspective of their parents behaviour at different ages. For example, young men may recall lower levels of care than middle-aged men due to a lack of life experience, negative feelings towards parents may be stronger, or they may have a tendency to over-report the degree of low care. Also in contrast to Parker, et al., (1979), the individual's gender does appear to influence the reporting of maternal or paternal care or overprotection in the current findings.

4.6 Summary

The hypothesised age-related differences in symptomatology were supported. However, some symptoms that were not expected to change with age were significantly associated with age. Anhedonia, psychosis, and functional impairment all declined with age when measured by self-report. It appears that self-report measures show that more symptoms decline with age than clinician rated scales indicating a possible bias in the results. It may be this bias that contributed to some of the mixed findings for anhedonia, psychosis, suicidal ideation, and social impairment within the current study, and the discrepancy between the results from the current study and previous findings. The hypothesis regarding Axis I comorbidity was supported. However, the hypothesis regarding axis II comorbidity was not supported because the regression models were not able to predict the presence of individual PD's, but, both, personality disorders and symptoms declined with age. Hypotheses regarding risk factors were both supported and not supported. Age-related changes in the character dimensions of personality were expected but the age-related changes in the temperament dimensions of personality had not been reported previously. The finding that CSA was not associated with age was expected. The mediating role of gender in the association between familial history and parental bonding was not expected, or the association between age and parental bonding.

4.7 Strengths and Limitations of the Present Study

There are a number of limitations in the present study which need to be considered when interpreting the results. Firstly, due to the cross-sectional nature of this study definitive

causal accounts (e.g., younger age causes greater suicidal proneness) cannot be made. It is not certain that the personality evaluation was not influenced by participant's depressive states (Luty, et al., 2002). In addition, some results may be due to a cohort effect. Given the reports of cohort effects for the rates of MDD and suicide such an effect is not unprecedented (see Wallace & Pfohl, 1995). Some of the current findings are comparable with research from the 1980's and 1990's. Yet, perhaps the differences seen in comparison to other studies are cohort effects. Therefore, while many correlations were found, causation remains unknown.

A further limitation is that it is difficult to compare some of these results to previous findings due to different symptom definitions (e.g., suicidal attempt or suicide ideation), different measures (e.g., SCID or DISC) different sample characteristics (e.g., inpatient or outpatient, cultural diversity, percentage women), different procedures (e.g., taking measures at baseline or during treatment) or reporting practices (e.g., self-report or interview). The fact that this thesis contradicted some previous work may be due to these factors.

Unfortunately, this study was unable to control for age at onset of depressive episode because current age and age at onset were highly correlated and not independent of each other. Hence the possible effect of age of onset on the findings is difficult to assess. In addition, duration of depressive episodes and number of episodes were not controlled for and may have contributed to the findings, particularly because recurrent depression is also associated with early-onset depression (Birmaher, et al., 1996). It is also unfortunate that adolescents were not included in the sample to allow for better comparison across development.

Due to sample characteristics and the study procedure these findings may not generalise to the population in general. Participants were predominantly of Pakeha/European descent and were not randomly selected. The individuals in this sample were referred for voluntary treatment, hence participants were help-seekers. Help-seeking may be associated with early-onset depression and positive family history (see Kendler, et al., 1999) and may have biased the sample. In addition, there may also be an age-related selection bias where, for example, young individuals with early insomnia are more likely to seek treatment than older individuals with early insomnia. Hence, these results may not generalize to all individuals experiencing a MDE. It is noteworthy that eight percent of the sample were diagnosed with

Bipolar II. However, it is unlikely that these findings would be significantly skewed by such a small subset of individuals.

Further limitations of this study arise from the statistical analyses. As mentioned above, the amount of variance accounted for in age by the dependent variables individually was often low. Therefore, strong statements cannot be made about the prediction of any of these dependent variables from age. In addition, multiple comparisons were made in the analyses, thereby increasing the likelihood of making a Type II error.

Some strengths of the study, however, were the large sample size and the use of a variety of measures. Semi-structured interviews, clinician-rated scales and self-report scales were used. All measures used show satisfactory reliability and validity. Research has demonstrated that that individuals with depression exhibit negative cognitive biases about the self and others (see Cicchetti & Toth, 1995). Hence, using multiple modes of measurement minimises bias in the current findings. Although, as suggested earlier, an age-related bias may have occurred on the different measures.

4.8 Implications

This study has covered some new ground and sets the stage for further studies to investigate differences between early and middle adulthood. Clearly, there appear to be developmental differences in the expression, comorbidity and the occurrence of identified risk factors for depression between early and middle adulthood. Some evidence showed similarities between young adulthood and adolescence. Therefore, there may be some evidence to support Arnett's (2000) theory of 'emerging adulthood' where young adults are distinctly different from middle-aged adults. Although the question of at which age adolescence ends has not been answered, this study has revealed some evidence for further research of this question. As suggested by previous research, the notion of 25 being a significant transition point for adulthood as opposed to 18 years challenges a number of studies claiming to study 'adults' when in fact those individuals have not yet emerged into adulthood. Therefore, further clarification of developmental changes is necessary to investigate if 25 is a significant transition point for individuals with depression, and also for individuals in other psychiatric populations and the general population. Other major issues in this area, which also require clarification, include whether the possible developmental

transition is influenced by cultural factors or gender, and whether these developmental changes occur in a linear fashion.

Another area that remains unknown is why these symptoms and the occurrence of identified risk factors change with age. Answers may be found through the investigation of stressful life events, socialisation processes, coping skills, emotional regulation, brain physiology, enhanced normal developmental changes, etc.

Further research with participants ranging from childhood to older adulthood would provide more sound evidence for developmental differences. This study was problematic because it could only compare findings with literature about adolescent depression where studies used different definitions, procedures, and had different sample characteristics. Therefore, longitudinal, life course studies are needed.

This study will hopefully encourage clinicians and other consumers to take caution when generalising results from college students/ young adult samples to adults as a whole. It should also encourage clinicians to be flexible with diagnostic criteria because obviously the criteria in the DSM-IV-TR (APA, 2000) for children and adolescents may extend to young adults. In addition, therapy needs to be tailored to the developmental differences in the expression of depression. Finally, it should encourage future research to investigate developmental differences between adults across a variety of psychiatric disorders because combining 'adults' into one bracket disguises critical differences.

4.9 Conclusion

This thesis illustrates that there appears to be significant age-related differences in the symptomatic expression of MDE, comorbidity and the occurrence of identified risk factors associated with depression in 431 outpatient young and middle-aged adults. Data also suggest that men and women may show differing patterns of age-related changes. Some of the findings in this study were mixed and some findings were contrary to previous research. However, it appears that this was often due to age-related discrepancies between self-report and clinician rated measures. This study has confirmed the necessity of researching age-related differences within adulthood, which may have substantial implications in the conceptualisation, assessment and treatment of adult depression. A number of variables, that were not controlled for in this study, may have contributed to the significant results such as

cohort effects, duration of depressive episode, age at onset or selection bias. Hence, future research needs to control for these variables to permit accurate conclusions.

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LIST OF APPENDICES

APPENDIX A

CHILDHOOD SEXUAL ABUSE INTERVIEW

E. CHILDHOOD SEXUAL ABUSE (ask in initial interview only, otherwise skip to Section F, p.14)

Sometimes traumatic events happen when people are quite young.

E.1 Did you experience any of these traumatic events before you were 16:

	Yes	No	D.K.
Were you the victim/witness of a disaster, accident or war, which affected your ability to live as before	1	2	9

	Number of times				
Before 16, were you ever:	0	1	2 - 3	4 +	D.K.
Threatened with abuse by someone	1	2	3	4	9
Emotionally or psychologically abused	1	2	3	4	9
Beaten so badly you had to see (or should have seen) a doctor	1	2	3	4	9
Other. Specify:	1	2	3	4	9

E.2 When you were under 16, were you ever physically or psychologically forced by anyone to engage in any unwanted sexual activity, such as unwanted sexual touching of your body or sexual intercourse?

Yes, definitely (Ask E.3)	1
Yes, perhaps (Ask E.3)	2
No, definitely	3
D.K.	9

DECISION: If 'no' go to Section F, p.14.
If 'yes' (perhaps or definitely) ask E.3

E.3 Did this involve: (If DK code 99; If >87 code 87) No. of times

Someone exposing the sex parts of their body to you when you didn't want it?			
Someone threatening to have sex with you when you didn't want it?			
Someone touching the sex parts of your body when you didn't want this?			
Someone trying to have sexual intercourse with you when you didn't want this and not succeeding			
Someone having sexual intercourse with you when you didn't want this			
Someone sexually attacking or raping you			
Other unwanted sexual activity. Specify:			

E.4 How old were you when this (these things) happened?
(Interviewer: For each year and each type of activity, enter code
Yes = 1
No = 2
N.A.= 8

Age	Exposure	Threaten	Touch	Intercourse	Other
0 - 4 years					
5 - 9 years					
10 - 12 years					
13 - 15 years					

E.5 What was the relationship between you and the person/people involved in these activities?

If 'yes' enter number of times it happened (before age 16)
If D.K. enter 9 but try for a best estimate
i.e. Was it 2-3 times or 8-9. If >9 enter 9

	Exposure	Threaten	Touch	Intercourse	Other
(Natural) father					
Brother(s)					
Uncle(s)					
Grandfather(s)					
Stepfather					
Stepbrother(s)					
Other rel. Specify					
Neighbour					
Family friend					
Teacher					
Stranger					
Other. Specify					

E.6 At the time, what were your reactions to this activity ?
Did you feel:

	Yes	No	N.A.	D.K.
Puzzled, confused	1	2	8	9
Distressed	1	2	8	9
Anxious	1	2	8	9
Frightened, afraid	1	2	8	9
Lonely	1	2	8	9
Embarassed	1	2	8	9
Ashamed	1	2	8	9
Angry	1	2	8	9
Other. Specify:	1	2	8	9

E.7 Do you think these events have caused you any continuing problems?
Specify:

Yes	1
No	2
N.A.	8
D.K.	9

E.8 Can you think back to the most serious or the worst episode(s) and describe it?
ie.the circumstances, involved, who was where it happened,what happened, your reactions.
(Interviewer: Record verbatim in as much detail as possible)

E.9 Have you ever told anyone else about any of this (these events)?
If yes, specify:

Yes	1
No	2
N.A.	8
D.K.	9

E.10 Have you ever received counselling for this/these events? (including those before or after you were 16)

Yes	1
No	2
N.A.	8
D.K.	9

IF YES: Ask E.11 - 12.

E.11 When did you last see someone for counselling?
(If current, code 00; If >97 months ago, enter 97;
If >97 months ago, enter 97;
If D.K. enter 99.

Months ago | | |

E.12 Who did you see and how many times?
Specify counsellor, service (name), and no.times seen.

(If >97 times, enter 97; If D.K. enter 99.)

Type of Counsellor	No. Times	
G.P.		
Psychiatrist		
Psychologist		
Other M.H.P. Specify:		
Other. Specify:		

APPENDIX B

FAMILY HISTORY INTERVIEW

FAMILY HISTORY SHEET: **I.D. No:** | | | | |

Interviewer: Complete one sheet for each parent, sibling or child (15 or over) with full biological relationship; and spouse/partner (current, at least 6 mths).

1. Name of relative: .....

2. Is subject's relative:

mother	1
father	2
daughter	3
son	4
sister	5
brother	6
spouse/partner (female)	7
spouse/partner (male)	8

3 a. Is relative still living? ..... Yes No

b. If **Yes**, go to Q.4.
If **No**, what was the cause of death? suicide (.....) 1
other (.....) 2

year of death 19____

4. History of Mental Illness:

Ask if relative ever had problems with any of the categories in A, then complete Section B where appropriate.

	A. Diagnosis Code: 1 = no, 2 = possible, 3 = definite) Go to B	**B.** For each 2 or 3 in Section A code Y(es) or N(o).		
		Caused social incapacity	Hospitalization for this illness	Treatment given e.g. somatic, psychotherapy
Major Depression				
Mania				
Alcohol Dependence				
Drug Dependence				
ASPD				
Eating Disorder				
Anxiety Disorder (Panic, OCD, Soc Pho)				
Other Psychiat. Dx. (.....)				
Other Dx. of note (.....)				

5. Family (1°) history of antidepressant treatment resistance:

Has 1° relative been treated with antidepressants ..... No (0) Yes (1)

Treatment response
Is there a family history of treatment resistance (ie ≥ 2 adequate trials)? ..... No (0) Yes (1)